ANNALS
OF
OTOLOGY, RHINOLOGY
AND
LARYNGOLOGY

VOL. 53 JUNE, 1944 No. 2

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CRITICAL REVIEW OF PATIENTS SUBJECTED TO LABYRINTH OPERATIONS

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A comprehensive symposium on labyrinthitis was presented before the American Otological Society in 1934. No useful purpose would be served in presenting a review of the literature at this time because it was so thoroughly done by Friesner and Rosenwasser.[1] Nothing of importance has appeared in the literature since. In that symposium general principles of the clinical and surgical management of the disease were discussed thoroughly. In presenting the results obtained in a group of cases of diffuse suppurative labyrinthitis in which operation on the labyrinth was instituted for what appeared to be logical reasons, I am not unmindful of the fact that no one observer's experience is sufficient for him to make dogmatic statements regarding the surgical treatment. The experience here reported may be of interest.

From the correlation of the clinical picture of labyrinthitis with histologic and anatomicopathologic observations the concensus seems to be that labyrinthitis may be classified into four groups: (1) circumscribed labyrinthitis, (2) diffuse serous labyrinthitis, (3) diffuse manifest suppurative labyrinthitis and (4) latent suppurative

From the Section on Otolaryngology and Rhinology, Mayo Clinic.

Read before the annual meeting of the American Otological Society, New York, New York, June 5-6, 1944.

labyrinthitis. However, the term "latent" may not be as meaningful in this respect as would the term "quiescent."

There is unanimity of opinion regarding the treatment of circumscribed and diffuse serous labyrinthitis. There is some difference of opinion regarding the treatment of diffuse manifest suppurative and latent suppurative labyrinthitis. In this connection I am reminded that Balance once said in respect to divergent opinion among competent observers that when once the pathologic process is understood there should be no divergence of opinion regarding treatment.

A question frequently asked is, when should operation on the labyrinth be employed. Why there should be any confusion in the minds of competent observers is hard for me to explain. In deciding the question one must be guided by his estimation of the symptoms and signs and of the probability of extension of the infection beyond the confines of the end-organ if it has not already taken place. The abdominal surgeon does not wait for peritonitis to develop before he removes an infected appendix. In any discussion about such a serious disease as suppurative labyrinthitis it should be emphasized that all efforts are bent on saving life. Clinical and surgical judgment, for which there is no substitute, and not statistical tables must decide what therapeutic measure is necessary in any given case. If an operative procedure is decided on, it would seem logical that labyrinthotomy, which is less severe than labyrinthectomy would be preferable to waiting until labyrinthectomy was indicated.

In general there are two types of operative intervention suitable for diffuse suppurative labyrinthitis. The first is labyrinthotomy. This type of operation depends on drainage for control of the disease. Operative procedures have been described by numerous surgeons, Hinsberg,[2] Kerrison, Richards and others. There is but little variation in the technics. This type of operation is suitable when there is no evidence of extension of the disease beyond the confines of the otic capsule.

The second type of operation is labyrinthectomy, or removal of the labyrinth together with purposeful wide exposure of the dura over the posterior and middle cranial fossae. This operation is indicated when there is evidence of extension of the infection beyond the confines of the otic capsule. A satisfactory operative procedure was described by Neumann.[3] If an abscess of the cerebellum is present the exposure afforded by the operation is adequate for drainage of the abscess.

There seems to be general agreement among competent observers that diffuse suppurative labyrinthitis occurring in cases of acute or subacute suppurative otitis media is more serious than the diffuse labyrinthitis occurring in cases of chronic suppurative otitis media. The debatable question in this situation is whether it is advisable to operate immediately and indiscriminately in those cases in which the labyrinthitis occurs in the course of an acute or subacute suppurative otitis media or to operate only when signs of extension of the infection to the intracranial structures occur. Hinsberg held that operation should be instituted early; that is not to say in the acute fulminating stage, however.

In the type of diffuse labyrinthitis which occurs after the radical mastoid operation owing to traumatic dislocation of the stapes or to too meticulous removal of the cancellous bone around the capsule of the labyrinth causing an irritative process within the canal or the static labyrinth, it seems to be the consensus that operation on the labyrinth is not urgent unless alarming signs occur. The differential diagnosis between diffuse serous and diffuse suppurative labyrinthitis may be difficult to make. The duration of the symptoms to me is a factor in deciding whether the process is serous or suppurative. The suppurative process destroys the function of the end-organ and the subjective symptoms may be relieved relatively soon. This is not so likely to occur in serous labyrinthitis. Operation would not be considered if the lesion is of the serous type.

In cases in which the posterior aspect of the petrous tip is involved by a suppurative process the function of the eighth nerve may have been destroyed not by primary involvement of the end-organ but by involvement of the nerve itself in the internal auditory canal. In such cases the finding of a nonfunctioning end-organ may be present but there is no history of prolonged labyrinthine reaction. This to me is extremely important information. I cannot conceive of any inflammation in the end-organ that did not cause severe incapacitating vertigo, nystagmus and nausea on any change of position of the head. However, when the eighth nerve is involved directly or primarily in the internal auditory canal the symptoms may have lasted but a few hours. Labyrinthectomy extended to the posterior aspect of the petrous tip is the most satisfactory approach in this instance.

In chronic suppurative otitis media and mastoiditis diffuse serous or suppurative labyrinthitis may follow circumscribed labyrinthitis, if the disease causing the circumscribed lesion is not eliminated.

If diffuse suppurative labyrinthitis occurs either before or after a radical mastoid operation for the control of circumscribed labyrinthitis, it is my opinion that the labyrinth should be opened at the optimal time because no further "danger signals" from the labyrinth can be expected to give warning of extension of infection to the intracranial structures until signs of meningitis or disease of the brain become manifest.

In the so-called latent type of labyrinthitis when chronic disease of the middle ear and mastoid is still present the findings at operation should decide whether operation should be performed on the labyrinth. If a large fistula or multiple fistulas were encountered either draining pus or not, I should prefer to be sure that adequate removal of the disease is carried out. The fear of awakening a dormant process can be overestimated. However, if there were no evidence of a break in the integrity of the membranous portion of the end-organ and there had been no recent symptoms and signs of recent activity of labyrinthine disease even though the end-organ had not responded to stimulation, opening into the labyrinth would not seem to be justified.

MATERIAL PRESENTED

The material for this report consists of the study of 12 cases in which labyrinthotomy was used for the control of diffuse suppurative labyrinthitis and 11 cases in which labyrinthectomy was used because the pre-operative observations or the operative findings seemed to indicate it to be the operation of choice. In Tables 1 and 2 the cases are listed in the order in which they were encountered beginning in 1917.

Labyrinthotomy.—In three of the 12 cases in which labyrinthotomy was performed the patients died (1,3,4). One patient died of meningitis; in addition, at necropsy generalized miliary tuberculosis was found. This was the first case in which I performed labyrinthotomy. Because at operation multiple fistulas and pachymeningitis were encountered, it is evident on the basis of present knowledge that labyrinthectomy should have been performed. There was no pre-operative evidence of disease of the central nervous system. Why the cerebellum was explored is not evident from the records. The two remaining patients died from meningitis due to an unrecognized abscess in the cerebellum at the cerebellopontine angle. Both of these deaths occurred late in the postoperative period. The results of the pre-operative neurologic examination had been reported negative. In Case 4, the cerebrospinal fluid was reported negative

pre-operatively. In this case, a radical operation was performed originally and circumscribed labyrinthitis was encountered. Twenty-four days later symptoms and signs of diffuse labyrinthitis developed and after observation for six days the labyrinth was opened because the patient was not making any headway. Labyrinthectomy would have been a better choice of operation than labyrinthotomy.

In Case 9, bilateral nonfunctioning labyrinths were encountered. Operation on the left mastoid revealed a fistula discharging pus. Labyrinthotomy was performed. One month later operation was performed on the right mastoid and no fistula was encountered. The labyrinth was not opened. This patient was observed at intervals for 15 years.

All patients had had neurologic and fundus examinations but apparently the spinal fluid was examined in Case 4 only. None of the patients were subjected to operation during the fulminating stage of the disease except the patient in Case 4. Three patients (2, 4, 9) were observed in the fulminating stage. All patients gave the history of having had a definite attack of labyrinthitis in the past. The time varied from those seen in the acute attack (2, 4, 9) to one (6) who had had an attack six years previously. The majority gave the history of an attack within a year. The chronic suppurative otitis media had been present in all cases for a long time. The patients ranged in age from 22 to 59 years. Cholesteatoma appeared to be the cause of the fistulas except in Case 10. In Case 7 the original attack followed the attempted removal of an aural polyp through the external auditory canal.

Facial palsy was encountered pre-operatively in three cases (8, 10, 12). The facial nerve did not recover its function in Case 10.

A most curious complication followed operation in Case 6. The jugular bulb was injured. Symptoms and signs of sinus thrombosis followed. It was expected that the thrombus would be found in the jugular bulb but the thrombus was found near the posterior knee of the sigmoid sinus.

If one can say that after six months labyrinthitis may be considered to be latent, then Cases 5, 6, 7, 8, 10 and 12 might be so classified. The pathologic changes found at operation decided the necessity for opening the labyrinth.

The mortality rate in this group was 25 per cent. The deaths occurred in the first four cases.

Labyrinthectomy.—In the 11 cases in which labyrinthectomy was performed there were no deaths. The patients ranged in age from 19 to 56 years. All the patients had had chronic suppurative otitis media and mastoiditis for long periods except the patient in Case 11. In this case the symptoms occurred during an attack of acute suppurative otitis. Curiously enough, in this group two patients (1,2) could not recall having had definite attacks of labyrinthitis although the tests revealed that no function was present. As a matter of fact, it was learned that the patient in Case 1 had been observed for six weeks and had been treated for typhoid fever, whereas actually he had had sinus thrombosis. Facial paralysis was encountered pre-operatively in three cases (6, 9, 10). The cerebrospinal fluid was examined pre-operatively in Cases 3, 5, 6, 9, 10 and 11. In all but Cases 9 and 10 pathologic changes were noted.

The initial attack began in Cases 3 and 4 while the patients were under observation for chronic suppurative otitis. Several patients were extremely sick (1, 2, 3, 4, 5, 6 and 11). Three patients (1, 3 and 11) were stuporous.

The operative findings were of particular interest. In Case 3 an abscess of the cerebellum, suspected pre-operatively, was found. The patient in Case 11 was known to have meningitis in addition to being six months pregnant. Caesarean section was performed before the mastoid operation.

Extensive pathologic findings were encountered at operation in Case 1; infected sinus thrombosis, epidural and subdural abscesses of the cerebellum in addition to the destruction of the otic capsule by cholesteatoma. Epidural abscesses were encountered in Cases 2 and 4.

Purposeful exposure of the dura revealed pathologic changes in Cases 1, 2, 3, 4, 5, 6, 8, 9 and 11. This, I think, is extremely interesting and perhaps very important. The question immediately arises as to whether or not purposeful exposure of the dura had not prevented further extension of the infection. Only in Case 11 were sulfonamide drugs used as adjuvant treatment. In fact, without the sulfonamide drugs, I doubt if this patient would have recovered.

In Case 3 the patient made a dramatic recovery after labyrinthectomy and drainage of the cerebellar abscess.

Facial paralysis occurred postoperatively but not immediately after recovery from anesthesia in Cases 1, 5, 6, 10 and 11. In all these cases the facial nerve recovered its function in varying lengths of time.

The dura under the epidural abscess in Case 4 was injured, causing the escape of cerebrospinal fluid, but this proved to be of no significance. The foregoing has happened to me several times in cases in which cholesteatoma was being removed from dura which had become very thin as the result of the overlying disease. In no instance has meningitis followed, even though the sulfonamide drugs were not in use at the time.

Of special interest were the clinical and surgical findings in Case 7. It was known that the symptoms were not due to diffuse labyrinthitis because the patient could hear. The operation revealed that the labyrinth had been destroyed by cholesteatoma but the cochlea had not been involved. Such a phenomenon is difficult to explain.

The greatest satisfaction came from the results obtained in Cases 1, 3 and 11.

In this group I could not classify any as latent labyrinthitis except Case 2.

SUMMARY AND CONCLUSIONS

In six of 12 cases in which labyrinthotomy was performed for the control of diffuse suppurative labyrinthitis, the pathologic condition possibly could be classified as the latent type of suppurative labyrinthitis. Three deaths occurred, one from diffuse suppurative meningitis and generalized miliary tuberculosis and two from meningitis following rupture of an unrecognized abscess of the cerebellum at the cerebellopontine angle.

There were no deaths in 11 cases in which labyrinthectomy was performed, either because of pre-operative conclusions or because of operative findings.

The observations and experiences presented in this group of cases would seem to justify the following conclusions:

1. Labyrinthotomy may be used with success in cases in which the history and signs of latent labyrinthitis are present, if, at operation, fistulas showing evidence of pathologic conditions in the labyrinth are encountered. Otherwise no intervention in the labyrinth seems justified.

2. Labyrinthotomy may be used with success in cases in which the history and findings of diffuse suppurative labyrinthitis are pres-

ent without signs of extension of the infection to the intracranial structures.

3. Labyrinthectomy may be used with success when the history and pre-operative observations indicate that there is definite activity of the infection in the labyrinth and that extension to the intracranial structures is imminent or has actually taken place.

4. To use labyrinthotomy when in reality labyrinthectomy is indicated is to court failure.

5. The optimal time for surgical intervention may depend on factors not readily reducible to definite statements.

MAYO CLINIC.

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TABLE 1

DATA ON CASES IN WHICH LABYRINTHOTOMY WAS PERFORMED

DURATION OF SYMPTOMS	CLINICAL FINDINGS	SURGICAL FINDINGS	COMMENT
	CASE 1 (Mr. T.) aged 33 yrs. 1917		
O.M.S.Ch.:* long. Labyrinthine: 6 weeks.	Nonfunctioning end-organ; headache; C. N. S. negative.	Canals and cochlear fistulas filled with granulations; pachymeningitis. Cerebellar exploration.	Died of meningitis; miliary tuberculosis.
	CASE 2 (Mrs. H.) aged 25 yrs. 1917		
O.M.S.Ch.:* long. Labyrinthine: acute, 3 days.	9-7-17: Hearing intact; too sick for vestibular tests; lay on rt. side; stupor; vertigo on motion. 9-27-17: Complete loss of hearing; not always nystagmus to rt.; nonfunctioning labyrinth; headache; C.N.S. negative.	Cholesteatoma in attic and antrum; fistula into cochlea draining pus.	
	CASE 3 (Mr. B.) aged 45 yrs. 1917		
O.M.S.Ch.:* long. Labyrinthine: 4 weeks.	Frontal headaches; severe vertigo at first; lately less; nonfunctioning end-organ; rt. fundus and C.N.S. negative.	Cholesteatoma; pus in cochlea and labyrinth.	Died; cerebellar abscess at angle; severe headache; sudden stupor.
	CASE 4 (Mr. B.) aged 40 yrs. 1917		
O.M.S.Ch.:* 40 years. Labyrinthine: 6 weeks.	Attacks of vertigo; copious discharge of foul pus; fundi and C.N.S. negative. Hearing intact. 10-25-18: Hearing gone; nonfunctioning labyrinth; severe headache; C.N.S., fundi and C.S.F. negative.	10-1-18: Fistula horizontal canal at radical operation. 11-1-18: Labyrinth opened; pus in canals and vestibule.	Died; severe pain in ear with negative C.N.S. findings until rupture of abcess at cerebellopontine angle with meningitis; facial paralysis.

TABLE 1—(*Continued*)

DURATION OF SYMPTOMS	CLINICAL FINDINGS	SURGICAL FINDINGS	COMMENT
	CASE 5 (Mr. C.) aged 28 yrs. 1920		
O.M.S.Ch.:* 9 months. Labyrinthine: 9 mos., lasted 1 week.	O.M.S.Ch.:* type IV with nonfunctioning end-organ; C.N.S. and fundi negative.	Fistula into horizontal and vertical canals draining pus.	Good result.
	CASE 6 (Mr. L.) aged 33 yrs. 1920		
O.M.S.Ch.:* 12 years. Labyrinthine: Several years previously.	Headache; foul discharge; increase vertigo; cholesteatoma; nonfunctioning end-organ; C.N.S. and fundi negative.	Large fistula horizontal canal draining pus; jugular bulb injured.	Sinus thrombosis in sigmoid sinus on 3rd postoperative day. Good result.
	CASE 7 (Mr. S.) aged 59 yrs. 1926		
O.M.S.Ch.:* 4 years. Labyrinthine: 1 year.	Removal of aural polyp followed by severe attacks with vomiting; nonfunctioning end-organ; O.M.S.Ch.* type IV; attic defect. Cholesteatoma; fundi and C.N.S. negative.	Cholesteatoma; fistula horizontal canal with polyp attached; free pus in labyrinth.	Good result.
	CASE 8 (Mrs. K.) aged 29 yrs. 1930		
O.M.S.Ch.:* Childhood. Labyrinthine: 1 year.	Vertigo for 2 wks., then none except on sudden movement of head; occasional generalized headache; facial palsy for 3 wks.; nonfunctioning end-organ; C.N.S. and fundi negative; O.M.S. Ch.* type IV.	Fistula into vestibule draining pus.	Good result.
	CASE 9 (Mr. C.) aged 32 yrs. 1931		
O.M.S.Ch.:* Childhood; bilateral. Labyrinthine: 8 days.	Sudden complete deafness with vertigo and vomiting; bilateral nonfunctioning end-organ; C.N.S. and fundi negative.	6 wks. later: fistula in left horizontal canal with pus. 1 mo. later: radical operation right; fistula but no pus; labyrinth not opened.	

TABLE 1—(*Continued*)

DURATION OF SYMPTOMS	CLINICAL FINDINGS	SURGICAL FINDINGS	COMMENT
	CASE 10 (Mr. T.) aged 22 yrs. 1930		
O.M.S.Ch.:* 8 years. Labyrinthine: ?	Rifle bullet middle ear; facial paralysis; chronic discharge; pain; occlusion of canal; nonfunctioning end-organ; C. N. S. and fundi negative.	Operation; bullet in capsule above horizontal canal; necrosis of bone throughout region.	
	CASE 11 (Mr. R.) aged 48 yrs. 1936		
O.M.S.Ch.:* 45 years. Labyrinthine: 6 months previously.	Severe vertigo lasting 2 wks.; occasional short attack of O.M.S.Ch.* type IV; nonfunctioning end-organ; C.N.S. and fundi negative.	Cholesteatoma; defect into cochlea by cholesteatoma.	
	CASE 12 (Mr. LeB.) aged 37 yrs. 1939		
O.M.S.Ch.:* 30 years.	Pain for 3 wks.; increased discharge, facial weakness; dizzy spells several years; nonfunctioning end-organ with facial palsy; C.N.S. negative.	Cholesteatoma with extension into labyrinth and around facial nerve.	Full recovery of facial palsy in 10 days.

*Chronic suppurative otitis media.

TABLE 2

DATA ON CASES IN WHICH LABYRINTHECTOMY WAS PERFORMED

DURATION OF SYMPTOMS	CLINICAL FINDINGS	SURGICAL FINDINGS	COMMENT
	CASE 1 (Mr. W.) aged 36 yrs. 1918		
O.M.S.Ch.:* 10 years. Labyrinthine: several weeks; no attacks recalled.	Pain, headache, chills, fever; had been treated for typhoid; small perforation; stiff neck, stupor, severely ill; mastoid tender; nonfunctioning labyrinth and cochlea. Temperature 101° F., P. 100, R. 22, W.B.C. 21,000.	Extensive sinus thrombosis; epidural and subdural abscess of cerebellum; cholesteatoma with erosion of otic capsule.	On 3rd postoperative day facial paresis developed. Almost complete recovery 4th week.
	CASE 2 (Mr. S.) aged 19 yrs. 1920		
O.M.S.Ch.:* 14 years. Labyrinthine: indefinite; no attacks recalled.	Exacerbation chronic ear. Pain; zygomatic abscess, concentric narrowing; nonfunctioning end-organ; C.N.S. and fundi negative.	Sclerotic mastoid: epidural abscess middle fossa; cholesteatoma in attic; fistula in horizontal canal draining pus; granulation in cochlea.	
	CASE 3 (Mr. C.) aged 30 yrs. 1921		
O.M.S.Ch.:* 2 years, right. Labyrinthine: left radical mastoid operation after 6 weeks' treatment (RAB)	25 days later: sharp pain; tender radical cavity; looked sick; no vertigo; no nystagmus; C.N.S. negative; ataxia. Severe headache 28th day. Vertigo, nystagmus, cerebellar signs left. Nonfunctioning end-organ 31 days—lethargy, stupor. C.S.F. 448 cells.	Exploration 31st day: cerebellar abscess; two fistulas horizontal canal draining pus.	Radical operation on rt. mastoid later.

TABLE 2—(*Continued*)

DURATION OF SYMPTOMS	CLINICAL FINDINGS	SURGICAL FINDINGS	COMMENT
	CASE 4 (Mr. McN.) aged 36 yrs. 1921		
O.M.S.Ch.:* 10 years. Labyrinthine: 5 days.	Lt. ear: foul pus; large superior perforation with attic defect; nonfunctioning end-organ; rotatory nystagmus to rt. Incapaciting vertigo. Hospitalized. Subjective relief after 18 days. Few months later returned. Nonfunctioning end-organ. C.N.S. and fundi negative.	Large fistula horizontal canal; epidural abscess at endolymphatic sac; spinal fluid escaped through thin dura.	
	CASE 5 (Mr. W.) aged 56 yrs. 1921		
O.M.S.Ch.:* childhood with acute flare-ups. Labyrinthine: 2 weeks, left ear.	Pain; severe vertigo; stiff neck; headache at night; frequent vomiting; rotatory nystagmus to rt.; nonfunctioning end-organ; C.S.F. cloudy, negative culture; fundi negative.	Sclerotic mastoid; cholesteatoma. Fistula in horizontal and vertical canals; pus in cochlea; red dura over sac; escape C.S.F.	Facial paresis lower part of face 3rd day.
	CASE 6 (Mr. R.) aged 39 yrs. 1922		
O.M.S.Ch.:* 15 years, left Labyrinthine: 2 weeks.	Headache; vomiting; vertigo lasted 1 week. Chills, fever; stiff neck; headache, worse at night; facial paresis; C.S.F. 1,600 cells, no bacteria; nonfunctioning end-organ; fundi negative. Progressively worse.	9th day; infected cholesteatoma. Granulations and large fistula horizontal canal. Dura over posterior fossa extremely red; face could be made to twitch from stimulation outer nerve. Endolymphatic sac destroyed.	Facial paresis, recovered.
	CASE 7 (Mr. B.) aged 49 yrs. 1934		
O.M.S.Ch.:* 25 to 30 years, bilateral. Labyrinthine: 2 weeks	Large polyp on left; small perforation on right; dizzy sign of fistula; rotatory nystagmus on looking up; hearing intact; C.N.S. and fundi negative.	Large cholesteatoma destroyed labyrinth; cochlea not disturbed because of hearing.	Retained hearing.

TABLE 2—(*Continued*)

DURATION OF SYMPTOMS	CLINICAL FINDINGS	SURGICAL FINDINGS	COMMENT
	CASE 8 (Mr. C.) aged 36 yrs. 1925		
O.M.S.Ch.:* 10 years, left. Labyrinthine: 3 months following influenza.	Vertigo lasted week or 10 days; pain since; aural polyp; foul discharge. Fundi and C.N.S. negative. Labyrinthine record missing.	Infected granulation; fistula horizontal canal draining pus; cerebellopontine angle exposed; dura red throughout.	
	CASE 9 (Mr. E.) aged 24 yrs. 1930		
O.M.S.Ch.:* 3 months. Labyrinthine: 18 days, right side.	Facial paralysis 18 days; vertigo, vomiting 18 days; deafness complete 18 days; no function of right labyrinth and left vertical canal; C.S.F. and fundi negative.	Infected granulation; fistula horizontal canal draining pus; cerebellopontine angle exposed; dura red throughout.	
	CASE 10 (Mrs. J.) aged 46 yrs. 1938		
O.M.S.Ch.:* 20 years; mastoid operation. Labyrinthine: 1 month.	Constant headache, worse at night; no vertigo; fistula sign—facial paralysis. Cochlea and labyrinth nonfunctioning; C.S.F. and fundi negative.	Antrum filled with granulations; destruction posterior aspect labyrinth extended deep into petrous, in fact surrounded pyramid; dura normal.	Facial paresis lasted several weeks.
	CASE 11 (Mrs. A.) aged 29 yrs. 1940		
O.M.S.Ch.:* 5½ weeks, left. Labyrinthine: from onset.	5½ months pregnant; hears low shout; surgical mastoid. Returned 5-19-40: Severe headache 5 days; nausea; vomiting; chills; stupor; stiff neck; Kernig et al; C.S.F. cloudy.	3-27-40: Complete mastoid; no fistula into labyrinth or cochlea made out. 5-20-40: Cesarean section; labyrinth draining pus at anterior end of horizontal canal; sequestrum surrounded facial nerve; dura red, exposed widely.	Recovery; facial paresis, recovery in 3 wks; chemotherapy.

*Chronic suppurative otitis media.

XXI

TEACHING OTOLARYNGOLOGY IN WARTIME

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In any specialty an essential prerequisite for instruction is a sound foundation of general medical training and experience. For this reason, otolaryngologists, as well as medical educators in general, have been keenly interested in the effect upon medical educational standards of war emergency measures primarily concerned with the increased production of doctors. Because wartime modifications in educational methods have influenced the teaching of otolaryngology they deserve careful examination.

The accelerated program for medical education exhibits many revolutionary trends and these are first apparent in premedical training. Here the most important educational experiment consists in the complete elimination of the economic requirement for the study of medicine since those to be educated are no longer dependent upon the ability to pay for their education. Furthermore, the Army and Navy College Training Programs have selected the students on merit alone and regardless of pressure or favoritism. Medical educators are agreed that the present group of premedical students equals or is superior to those selected under the former system.

By subsidizing superior students and those with limited financial resources, by providing undivided time and energy for study, and by selecting students for the study of medicine at an earlier academic period than before the war, medical education may actually be benefited. Some features of acceleration in premedical training, however, have not received general approval. Thus, the elimination of the long summer vacations, increasing the daily and weekly assignments, reduction in elective work and alterations in curriculum continue to be regarded with suspicion.

The Army and Navy are selecting 90 per cent of the medical students. While medical faculties continue to choose the students for undergraduate medical instruction, bilateral selection by the school and the student has been eliminated. Upon selection, the

From the Transactions of the American Laryngological Association, 1944.

student is placed in a pool and neither the school nor the student determine which school shall be attended. After January 1, 1945, the incoming freshman classes will have had only the concentrated military courses.

When, at the onset of hostilities, the medical schools of the nation were confronted by a program entailing a 20 per cent increase in classes, a reduction of the four-year course to three years, and the release of up to 50 per cent of their teachers to the armed forces, it was obvious that a tremendous task lay ahead. In spite of continuous session, modified courses, shortened hospital training and the absence of many former teachers in the services, there has been a determined effort to keep up the quality of the work.

Continuous session has been criticized because it endangered the health of medical students, seriously lowered medical educational standards, prevented orientation of the student, necessitated large classes with minimal personal instruction and fostered an abbreviated curriculum. Whether regimentation will affect the health of the students remains to be determined. The other points of criticism can be profitably examined in the light of the past two years' experience.

In otolaryngology, there has been a definite lowering of educational standards. The deterioration in personal instruction has resulted from depletion of the faculties, however, rather than because of large classes. Curricular changes have, as a rule, not been great, and in some instances the curriculum has actually been augmented. Upon one point there can be no difference of opinion; the accelerated program is definitely increasing the production of doctors.

The clinical faculties in a high percentage of the medical schools of the United States are composed of volunteers. This applies particularly to pedagogy in otolaryngology and other specialties. These faculties were depleted of their younger and most active teachers early in the war. The highly specialized forms of teaching were the ones most affected, and they are continually losing more men formerly regarded as holding key positions. They cannot be replaced. Moreover, the ranks of the older teachers in our specialty are being constantly thinned by death and infirmity, and at a rate materially increased by the combined burden of an increased teaching schedule and increased private practice.

Clinical instruction in otolaryngology requires teaching material. Serious paucity of such material has resulted from economic improvement in the lower wage groups and ready employment at

high wages. The out-patient departments of many of our teaching hospitals are almost deserted. Continuous session, requiring dispensary teaching in the summer months when acute infections of the ear and upper respiratory tract are at a minimum, still further limits clinical instructions.

In spite of these handicaps, undergraduate instruction in otolaryngology compares favorably with that of prewar sessions. Because the object of undergraduate instruction and internship is to equip the individual for the general practice of medicine and not for direct entry to any specialty, the imminence of military service has affected the curriculum very little. If to an already crowded curriculum anything is to be added, certainly nothing in the nature of broad fundamental training should be dropped.

Since younger medical officers have shown deficient training in certain directions the following suggestions are made. There is need for emphasis on hearing tests, malingering tests and the interpretation of audiometer findings. The contributions of aviation medicine to otology are neglected. There is not sufficient attention to the subject of acoustic trauma which, because of the large number of blast injuries, has become of increasing importance. Tropical warfare has demonstrated again that diphtheria is endemic to the tropics and that mycotic and fungoid diseases can reduce the fighting strength of our forces. Instruction in the management of these diseases should be specific and adequate.

The armed forces regard the recent graduate as most desirable, and from this group intern-residents must be chosen. Even in peace time there were more approved internships than eligible candidates. To solve the conflicting interests of military necessity and civilian medical services, two plans were adopted. The 9-9-9 intern-resident program applies to both militarized and non-militarized personnel in fixing the period of appointments as hospital personnel. It applies to militarized personnel only in the limitation of appointment to one period each as intern, assistant resident and resident. Those not subject to military orders continue to be eligible for prolonged training. Actually, this has afforded two-thirds the former number of interns, one third of whom are deferred to serve as junior residents, and one-half of the assistant residents may be deferred for a third nine-month period as residents. The second plan, the hospital quota program, allots to each hospital a quota made up of both military and non-military house officers.

In each crop of medical students receiving training under this system, several hundred who would normally continue their prepara-

tion for careers in medical teaching and research are lost. This is of grave concern, for from this group future leaders in the specialties would normally emerge. There is no provision for the training of a continuous flow of teaching personnel. To minimize a serious permanent loss to medical education, those young men of ability, interested in academic careers, must be returned promptly after the war and every facility provided for their development.

The prospective specialist in otolaryngology should first narrow his interests to his chosen field during service as an assistant resident or resident. It is this phase of instruction which has suffered most.

Regardless of whether the internship is rotating, mixed or straight, the reduction of the internship to nine months lowers educational standards because the total time for patient contacts is reduced. In addition, the depletion of medical and nursing staffs and the dearth of hospital employees make it impossible to impress upon the interns the thoroughness and attention to details essential for the development of sound professional habits. Observation of recent graduates in military service reveals that training in medicine and general surgery has been adequate, but at the expense of instruction in otolaryngology and the other specialties.

The various services have appreciated the need of specialized training for medical officers. This has been provided to some extent by advanced instruction here and abroad. Such instruction, however, is designed to meet wartime problems and not to make specialists. The larger military hospitals hold regular conferences and staff meetings and service hospitals adjacent to medical schools provide medical officers for special clinical instruction to students. Members of medical faculties are appreciated lecturers before service hospital staffs. None of these represent postgraduate instruction in an acceptable form. Because of the tempo of the war, the rapid expansion of the medical corps, and the availability of reputable and well-trained specialists for supervisory posts, postgraduate training is not of immediate importance to military authorities.

One extensive movement in graduate medical education, Wartime Graduate Medical Meetings, has been in operation for a year and a half. Under the auspices of the American Medical Association, the American College of Surgeons and the American College of Physicians, and with the authorization of the three surgeons general, instruction has been carried out in service hospitals throughout the country. Aided by the deans and faculties of 52 medical schools,

facilities have been provided for ward rounds, clinicopathologic conferences, study groups and practical demonstrations. The national faculty of this organization consists largely of the Fellows of the American Otological Society and the American Laryngological Association and other internationally known leaders in this specialty.

Clinical conferences and study groups in the larger medical centers are jointly planned by service hospital authorities, medical school faculties and regional committees of Wartime Graduate Medical Meetings. The ensuing discussions have benefited those in civilian practice and those in active teaching positions even though the primary object has been continuous graduate education to meet the needs of physicians in military service. There has followed a better understanding and appreciation of new clinical problems arising from the war.

Postgraduate education in the pre-war sense is a diminishing entity. Even when such training is obtainable, civilian physicians find it impossible to embark upon a one- to three-year course of special training. The short brush-up course, while neither producing specialists nor serving as a short cut to specialization, offers the only opportunity to obtain the stimulus and training afforded by contact with medical leaders. Such programs for small groups have been conducted by the Army, Navy, Public Health Service and the American College of Surgeons. It is well within the province of the national otolaryngological societies to develop, operate and direct short courses for small groups. Through their membership all the important clinical facilities in the United States are available. As a temporary measure until the end of the war, this type of instruction will in part compensate for the diminishing availability of organized postgraduate teaching.

In the specialties there is need for thought and planning for the postwar interval. Postwar educational facilities for returning medical officers must be provided if the best interests of the public and the profession are to be served. Those returning to hospital duties will succeed in obtaining adequate supervision and instruction but those, who of necessity, return directly to private practice will need and demand a thorough review of recent developments in otolaryngology or basic instruction in the specialty. The deans and faculties of medical schools should prepare to put into effect immediately upon the cessation of hostilities a plan to provide short courses of two to eight weeks' duration in which concentrated and practical instruction will be provided. The Council on Medical Education and Hos-

pitals of the American Medical Association is cognizant of these problems and has actively engaged in formulating plans which cannot be completed until the rate of demobilization of medical officers can be estimated with reasonable accuracy.

Those engaged in medical pedagogy must formulate positive plans not only for the duration of the war but for the postwar period and its difficult readjustments.

SANTA MARGARITA RANCH.

XXII

THE LOCAL USE OF SULFADIAZINE SOLUTION, RADON, TYROTHRICIN AND PENICILLIN IN OTOLARYNGOLOGY

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Ear and sinus disorders are a frequent cause of disability in our armed forces, particularly among aviators, submarine crews and deep sea divers, who are subjected to frequent and severe changes in temperature and barometric pressure. It has been observed that many of our men in the tropics as well as those in colder and damper climates develop excessive hyperplasia of lymphoid tissue in their pharynx and nasopharynx. This condition in adults as in children predisposes to frequent colds, chronic nasal congestion and postnasal discharge. In those subjected to great changes in pressure in the air or in the depths of the sea, the ear and sinus lesions may be purely mechanical. These lesions are designated as aero-otitis or aero-sinusitis in American literature but a better descriptive word, used by the British, is barotrauma. In the presence of an acute or chronic infection in the upper air passages these pressure changes often lead to suppuration, which necessitates hospitalization and often discharge from service.

From years of experience in treating children with somewhat similar conditions, we believe these men can be greatly helped by two simple and practical methods of treatment, local application of a sulfadiazine solution in the nose and nasopharynx to discourage the growth of bacteria, and beta and gamma irradiation of the pharynx and nasopharynx to remove hyperplastic tissue. The combination of sulfadiazine and irradiation is often better than either of them alone.

The solution we use for the treatment of an acute upper respiratory infection is 2.5 percent sulfadiazine in ethanolamines (Pickrell's

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From the Department of Otolaryngology, Johns Hopkins University and Hospital.

Read before the Sixty-Sixth Annual Meeting of the American Laryngological Association, New York, N. Y., June 8, 1944.

solution). The patient is instructed to put one-half medicine dropper of the solution into each nostril and allow it to flow backward along the floor of the nose into the nasopharynx and pharynx.

This should be repeated ten to twelve times a day for three days and four to six times a day for an additional week. Most of the drug administered in this way is swallowed and is of little value, since the blood level rarely exceeds 1 mg. per hundred cc. after a full course of this treatment. With each application, however, some of the sulfadiazine solution is absorbed by the lymphoid tissue and, judging from clinical and bacteriologic studies previously reported[1, 2] the concentration of the drug in the nasopharyngeal lymphoid tissue soon reaches a level that inhibits the growth of pyogenic bacteria.

We have used this method of treatment for prevention of ear and sinus complications of acute colds in approximately 1500 patients during the past three years, with results far better than we have obtained with any other form of treatment. Until the concentration in nasopharyngeal lymphoid tissue has reached a level that inhibits the growth of bacteria, the treatment is of no value. Therefore, it is necessary to begin the local application of this drug as soon as possible after the first symptoms of a cold and use it in large amounts and at frequent intervals for at least a week. This treatment continued for a month instead of a week, is often very helpful in chronic nasopharyngitis, if the infecting oganism is sensitive to the sulfonamides. Local irritation of the skin around the external nares may be avoided by applying cold cream. In a few patients with sensitive, allergic mucous membranes the sulfadiazine solution causes an increased feeling of rawness, burning and congestion. If this happens the drug should be discontinued, but in our experience less than five percent have any symptoms of allergic rhinitis or any evidence of sensitivity to the drug.

The sulfadiazine treatment gives only temporary relief. The real problem is to get rid of hyperplastic lymphoid tissue, which often extends into the posterior nares, grows up on the anterior wall of the sphenoid, on the lateral walls of the nasopharynx, around and in the orifice of the eustachian tubes and in other locations that are inaccessible for surgical removal.[3] It so happens that next to the sex cells, lymphocytes are the most sensitive cells in the body to beta and gamma irradiation. Therefore, in treating these patients the dosage employed is so small that there is no danger of a burn or a dry nasopharynx in which crusts form. The action of the irradiation is to inhibit mitosis in the germinal centers and thus stop the formation

of new lymphocytes. Observation of hyperplastic lymphoid tissue under this treatment leads us to believe that lymphocytes, like the skin cells, have a brief life cycle, probably not more than two weeks. Under irradiation treatment no new lymphocytes are formed to replace those discarded, and the mass gradually shrinks and finally disappears, leaving the nasopharynx covered with smooth mucous membrane not unlike that on the nasal septum. If the dosage is just right the result is perfect and there is never any recurrence. If the dosage is too great ecchymoses appear in the nasopharyngeal mucous membrane, but there is no excuse for ever getting a burn.

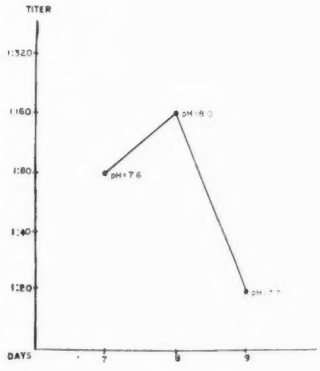
At the Johns Hopkins Hospital we use from 800 to 1000 millicuries of radon[4] with a brass filter 1 mm. in thickness, but a more practical applicator for use in the army and in civilian hospitals and office practice contains 50 mg. of anhydrous radium sulphate. The applicator is made of monel metal. The handle is 15 cm. in length and the radium-containing chamber 15 mm. in length, 1.5 mm. in diameter and 0.3 mm. in thickness. This allows the passage of more beta rays than the 1 mm. of brass in the radon applicator, but has been used with success in the Hagerstown Clinic for the Prevention of Deafness in Children, which is sponsored by the Maryland State Board of Health and the Children's Bureau in Washington. The results[5] in 259 patients have been as good as those obtained with radon, and the time required for each treatment is only 6.6 minutes. Of the entire group under treatment in Hagerstown the hearing has improved as much as 40 db. in 39 ears and 10 db. or more for several frequencies in 127. Fifteen chronically discharging ears are now dry and 15 patients with a history of repeated attacks of acute otitis media have been well since the irradiation treatment was finished. Thirty-two patients treated for severe recurrent upper respiratory infections have had very minor colds during the past winter. Ten patients with infected adenoids had severe asthmatic bronchitis with each cold; five of these have had no attacks during the past winter and the other five are greatly improved, a better result than we have ever obtained from operative removal of tonsils and adenoids in children.

Irradiation treatments should never be given until there has been a thorough inspection with a nasopharyngoscope of the nasal passages, the region of the orifices of the accessory nasal sinuses and particularly the nasopharynx and orifice of the eustachian tubes. The teeth, pharynx, tonsils and base of the tongue must also be examined. Palpation with the finger or mirror examination of the nasopharynx are not satisfactory, especially in children. It is not the size, but the location of lymphoid nodules that is important. Irradiation treat-

ments should be spaced at intervals of four to five weeks, and the nasopharynx must be examined with the nasopharyngoscope before each treatment. Greatly enlarged adenoids are better treated by operative removal, supplemented by irradiation at the conclusion of the operation. Often only one treatment is necessary, but the nasopharynx should always be examined again six months later and the irradiation repeated if there are any symptoms or visual evidence of recurrence.

At the Johns Hopkins Hospital we treat approximately 125 patients each month in the out-patient department with beta and gamma irradiation of the nasopharynx. This leaves our beds free for seriously ill patients and at the same time we obtain most satisfactory results in a group of patients who in pre-irradiation days would have been admitted for operative removal of tonsils and adenoids. For 15 years it has been our practice to irradiate the nasopharynx only, since treatment of the tonsils and pharynx would subject the doctor to too much exposure. If the tonsils are chronically infected they should be removed, but it is our impression from observation on several thousand patients that the nasopharynx is usually the primary focus from which infection spreads to the sinuses, ears and tonsils. If the nasopharynx is freed of lymphoid tissue, recurrent colds become less frequent and severe, and infections in the sinuses, ears and tonsils often heal spontaneously.

In giving irradiation treatments the best protection for the operator is a distance of 30 feet from the applicator. Two pairs of thin rubber gloves will protect the hands from beta rays while handling the applicator, but the use of a lead containing apron or gloves, as used in x-ray work, is of no value in protection against gamma rays. When not in use the applicator should be kept in a 7/16 inch hole in the center of a lead cylinder which is from four to six inches in diameter, depending on the amount of radium or radon in the applicator. This hole contains a glass tube filled with alcohol. Before inserting the applicator into the patient's nose, the excess alcohol should be washed off and the applicator dipped into boro-glycerine. This prevents nasal secretions from sticking to the applicator and is important because the hands must never be used to wipe the applicator. The first evidence of too much exposure of the hands shows in the nails, which become cracked and ridged. It is also wise to have a complete blood examination at intervals, but with care there is no danger, as evidenced by the fact that many of us have given these treatments several times a week since 1928.



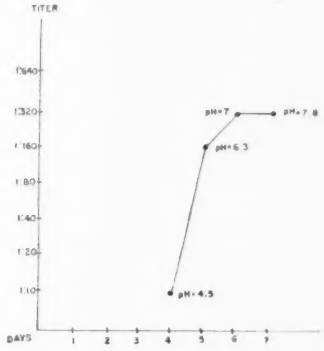


Fig. 1.—Changes in bactericidal titer and pH during growth of Penicillium. (From Fisher).

Fig. 2.—Shows the daily bactericidal titer of penicillin during growth, correlated with changes in pH. (From Fisher).

TYROTHRICIN

Tyrothricin is in many ways superior to either penicillin or the sulfonamide powders for local application. When it is brought into contact with a sensitive organism it kills almost instantaneously. It is truly bactericidal, while penicillin and the sulfonamides are bacteriostatic and must be in contact with the bacteria for several hours in order to produce a lasting effect. For this reason it is better to use tyrothricin to irrigate an acutely infected antrum, to fill the canal following paracentesis and to wash the wound and surrounding soft parts after every sinus and mastoid operation. If suppuration continues, it means the bacteria have not been reached by the drug, or they are resistent to the action of the drug. Both tyrothricin and penicillin when used locally either kill or inhibit most strains of staphylococci, streptococci (both aerobic and anaerobic) and pneumococci, but have no effect on Escherichia coli, Proteus, Pseudomonas aeruginosa (pyocyaneus) and Friedlander's bacillus. Tyrothricin is insoluble in water and does not penetrate tissues when applied locally. It is not only useless, but extremely toxic when injected into tissues or given intravenously. Penicillin on the other hand penetrates tissues well and for this reason is of value in the local treatment of chronic infections, provided the concentration in the wound can be kept at the proper level over a sufficient length of time.

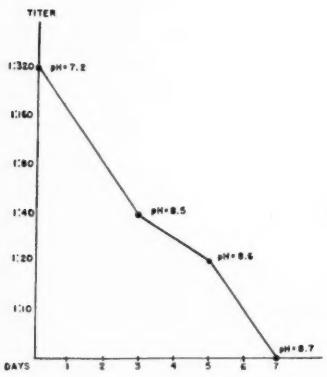


Fig. 3.—Shows the rapid loss of antibacterial activity when the filtrate is kept at room temperature. (From Fisher).

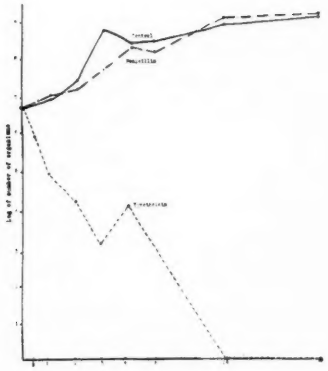


Fig. 4.—Shows how completely resistant a strain of staphylococcus aureus may be to penicillin, and how the same strain is sensitive to tyrothricin.

PENICILLIN

Crude penicillin[6] can be made easily and inexpensively by inoculating suitable liquid culture media with spores from stock cultures of Penicillium notatum, and keeping the inoculated flasks at room temperature (22° to 25° C.) until pH and titration tests indicate the antibacterial activity has reached its peak. The pH of the culture medium which starts at 3.5 to 4.5 is unchanged for three or four days, then rises rapidly. When the antibacterial activity also reaches its peak the pH is usually between 7 and 7.5, although this figure may vary with each lot. If the alkalinity is allowed to go much above this point the antibacterial activity of the medium is rapidly lost. Fig. 1 shows the changes in bacterial titer and pH during growth of the mold penicillium. On the eighth day after inoculation the pH of the fluid under the layer of growing mold is 8.0. The titer, (determined by adding this fluid to a series of broth tubes in the proportion of 1:20 to 1:320 and inoculating with a stock culture of staphylococcus) was 1:80 on the seventh day, 1:160 on the eighth day, but suddenly and rapidly fell to 1:20 on the ninth day. Contaminating organisms introduced into the flask at the time of inoculation with the mold, or when testing for antibacterial activity, will also inhibit the growth of the mold or destroy the activity of the penicillin that has been produced. To ob-

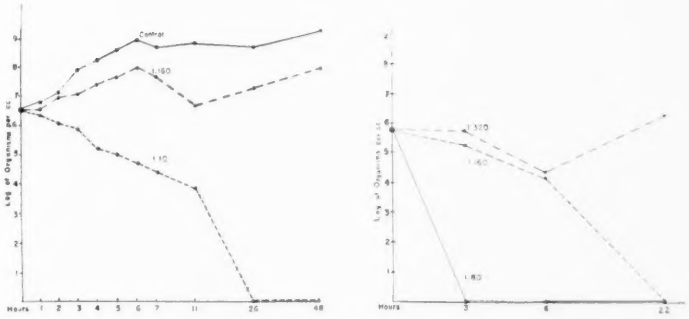


Fig. 5.—Both the concentration of the antibacterial agent and the time factor are important, as is shown in Figs. 5 to 8. Fig. 5 shows the effect of two dilutions of penicillin on Staphylococcus aureus, compared with the normal growth curve.

Fig. 6.—Shows the bactericidal and inhibiting action of three dilutions of penicillin on beta hemolytic streptococcus (group A). (From Fisher).

tain the most potent penicillin the contents of the flask must be passed through a Seitz filter at the right moment. Fig. 2 shows the daily bactericidal titer correlated with changes in pH.

Both crude penicillin and the purified sodium or calcium salt of penicillin are unstable. To maintain its potency crude penicillin is best kept in a -30° C. refrigerator. Fig. 3 shows the rapid loss of antibacterial activity when the filtrate is kept at room temperature. When needed for a treatment or operation, the required amount is thawed by placing the flask of frozen penicillin in warm water. The stock solution may be melted and refrozen repeatedly for as long as two months without loss of potency, provided it does not become contaminated. The most important points for maintaining the potency of penicillin are: adjust its pH close to neutral, keep it frozen and at all times completely free from contaminating organisms.

DISCUSSION

Many new and possibly better drugs are sure to be found through the intensive studies being made of the chemical composition of penicillin and similar substances, just as the various sulfonamides were developed following the demonstration of the antibacterial properties of sulfanilimide. Treatment with these drugs is of little value however, if the organism happens to be a resistant

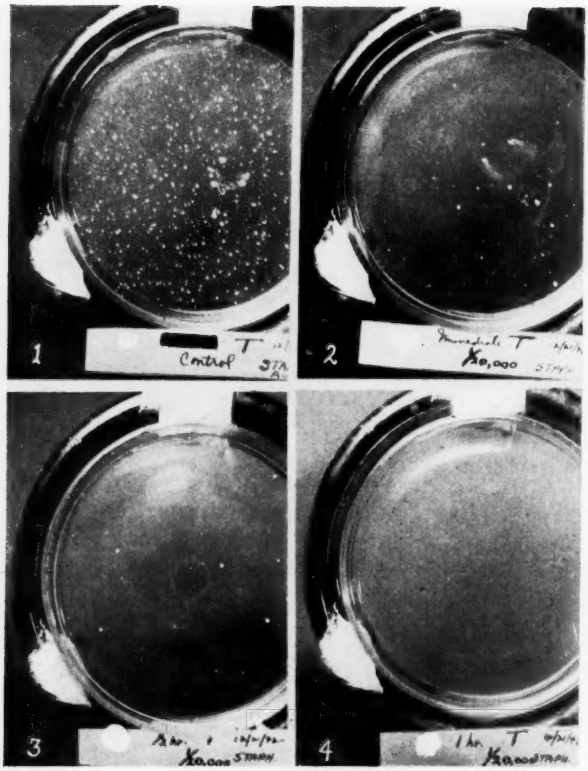


Fig. 7.—These photographs (1 to 4) show the rapidity with which tyrothricin kills staphylococcus, in contrast to the slower action of penicillin, shown in Fig. 8. The plate poured immediately, (i. e. within 2 or 3 minutes after inoculating with one drop of 24-hour staphycocccus culture a 5 cc. broth tube containing tyrothricin in the proportion of 1:20,000) shows a marked reduction in the number of organisms (2). The plate poured at the end of an hour is sterile (4).

strain. Careful and detailed bacteriologic studies are essential if the therapy of bacterial infections is to be placed on a rational rather than an empirical basis. The mere identification of organisms is not sufficient, especially if we are dealing with a staphylococcus aureus infection. Penicillin is useful in the treatment of staphylococcus infections, but of 90 strains of staphylococci (aureus and albus) Fisher[6] found by titration tests 12 that were resistant to penicillin. In addition, some strains of staphylococci in local infections acquire resistance to penicillin during the course of treatment.[7]

The organisms found most commonly in acute infections of the upper air passages are beta hemolytic streptococci, pneumococci, anaerobic or micro-aerophilic streptococci and H. influenzae. All of these are sensitive to penicillin and tyrothricin except H. influenzae. In addition to one or more of these sensitive organisms, resistant bacteria such as Escherichia coli, Proteus, Pseudomonas aeruginosa (pyocyaneus), Friedlander's bacillus and certain strains of staphylococcus aureus frequently are found in chronic sinus and ear infections. Of this latter group staphylococcus is the only organism that may be sensitive to penicillin, tyrothricin and the sulfonamides, while Escherichia coli is affected only by the sulfonamides.

When any chemotherapeutic agent is to be employed in the treatment of an infected patient it is important to identify as quickly and as accurately as possible the organism or organisms we have to deal with, and by titration tests determine their sensitivity to the various drugs at our disposal. After two years' experience we believe these test tube determinations of the sensitivity or resistance to penicillin, tyrothricin or the sulfonamides are a reliable indication of the result we may expect in the treatment of clinical infection with these drugs. Fig. 4 shows how completely resistant a strain of staphylococcus aureus may be to penicillin. If this happens in the test tube where the penicillin is in direct contact with the bacteria, it is evident that this drug could be of no value in the treatment of infection with this particular strain of staphylococcus.

One essential then for the successful treatment of infection with local chemotherapy is identification of the organisms and determination of their sensitivity. At this stage in the development of local chemotherapy this should be a part of the routine pre-operative examination of every patient. A second essential is to administer the antibacterial agent in such a way that it comes into actual contact with the infecting bacteria in effective concentration and for an

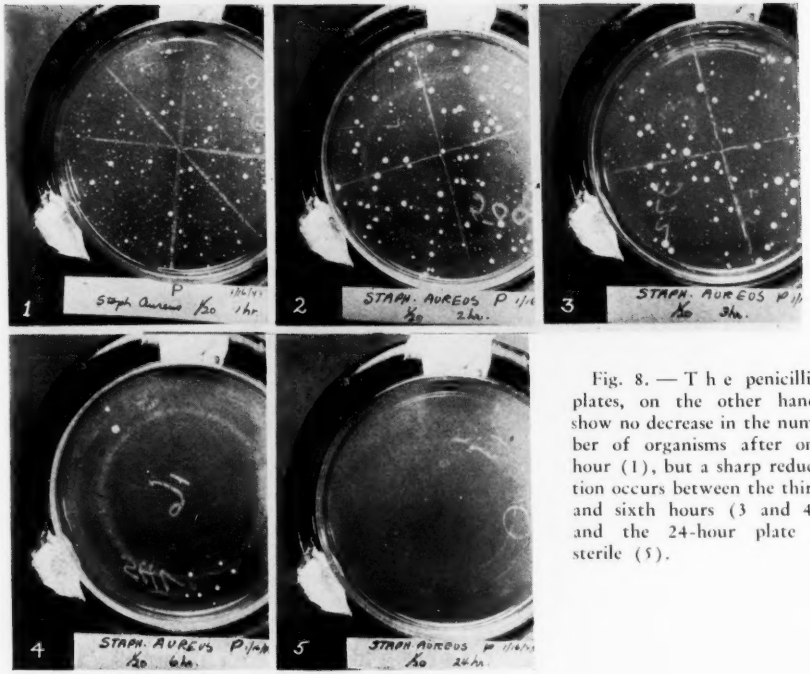


Fig. 8. — The penicillin plates, on the other hand, show no decrease in the number of organisms after one hour (1), but a sharp reduction occurs between the third and sixth hours (3 and 4) and the 24-hour plate is sterile (5).

adequate period of time. Both the concentration and the time factor are important as shown in Figs. 5 to 8.

Clinical experience shows that all staphylococcus infections do not respond promptly or satisfactorily to penicillin, even when given intravenously and in adequate amounts. Acute spreading infections are cured more easily than chronic infections especially when there are thick fibrous walls, a poor blood supply and sluggish drainage. This is equally true when penicillin, tyrothricin or the sulfonamides are applied locally in the treatment of infection. The rapid cure that often follows one irrigation of an acute frontal or maxillary sinus infection with crude penicillin or tyrothricin suggests that during the acute stage the organisms are superficial and the drug comes into contact with them, while in chronic infections the bacteria are located deep in the tissues. The reason irrigations are less effective in chronic infection is the difficulty of bringing the drug into direct contact with bacteria deep in the tissues. This difficulty can be obviated in chronic infections of the sinuses or mastoid by combining surgical operation for the removal of infected tissue and local chemotherapy to prevent wound infection. At the conclusion of the operation the bacteria are on the surface of the exposed bone and surrounding soft tissues, and are just as susceptible to the action of penicillin, tyrothricin or a sulfonamide powder as they would be in a test tube. If we know what organisms we are dealing with and use the drug or combination of drugs that will kill them without injuring the tissues or causing toxic symptoms, oft n it is possibe so nearly to sterilize a wound that postoperative fever, swelling, pain and suppuration are entirely absent. This means much for the comfort of the patient, shortens the period of hospitalization and saves time and energy for the surgeon.

Sensitive bacteria are killed within an hour by tyrothricin, but the action of penicillin (1:20) is at least six times as long. For this reason tyrothricin is preferable in an operation where it is important to reduce wound infection and its consequence to a minimum. If the infecting organism is sensitive to both tyrothricin and penicillin, the best results are obtained by using tyrothricin at the operation and crude penicillin, diluted with an equal amount of normal saline, to saturate the packs and for any postoperative irrigations that may be necessary. All antibacterial substances, including the sulfonamides, must reach the bacteria in adequate concentration in order to be effective. Tyrothricin is insoluble in water and kills only the bacteria on the surface of the wound, while penicillin penetrates and reaches bacteria in the tissues.

The absence of wound infection greatly reduces pain, swelling, suppuration and the frequency of postoperative treatments. Most important of all, prevention of wound infection reduces postoperative polyp and granulation-tissue formation aind thus makes it possible to cure, without a deforming operation, selected cases with a chronic frontal sinus infection. The frontal sinus may be entered through an exteranl incision in the eyebrow not more than 2 or 3 cm. in length. All infected mucous membrane together with the entire floor of the frontal sinus and all orbital extensions of the ethmoidal cells are removed and a large opening made into the nose. The wound and surrounding soft parts are thoroughly washed with 1:1000 tyrothricin and the frontal sinus packed with gauze saturated with penicillin. The end of the pack is brought out through the nose and the external incision closed. There is rarely any postoperative edema of the eyelid and the incision heals per primam. The pack is removed under light vinethene anesthesia 24 to 48 hours later. The frontal sinus is rendered so nearly sterile by this procedure that excessive growth of granulations is prevented and therefore the opening into the nose does not close, provided the operation has been thorough and the sinus is irrigated daily for ten days with either tyrothricin or penicillin. The choice of the best drug to use is determined by titration tests. After the first ten days, even though no gross pus has been seen in the irrigating fluid at any time since operation, the frontal sinus, in these non-deforming operations, should be irrigated with penicillin or tyrothricin at intervals for another two weeks. The object of these prolonged irrigations is to prevent the multiplication of any bacteria in the wound that may still be viable.

The following case history is of interest from the point of view of both the general and the local use of chemotherapy. A child was admitted to the hospital with a septic temperature and a tender swelling under the arch of the zygoma and the temporal muscle. There was no discharge from the ear, no swelling or tenderness over the mastoid and his hearing was good. The illness began with an acute nasopharyngitis two weeks previously. The family physician had given enough sulfathiazole by mouth to mask the symptoms of otitis media and mastoiditis, but not enough to kill the organisms. On admission he was toxic, dehydrated, and with the exception of the swelling in his temporal fossa his symptoms might have been due to drug fever. A prominent internist recently stated that in approximately half of the patients he had seen in consultation during the past two years, the symptoms were due to toxic manifestations of sulfonamide therapy. A wise rule is

to redouble the search for signs and symptoms of sulfonamide poisoning whenever therapy has been continued for longer than ten days.

In this child, however, both the location of the swelling and the mastoid films suggested a rupture of infected zygomatic cells and an abscess under the temporal muscle. At operation extensive destruction of the deeper cells in the mastoid process and an osteomyelitis of the zygoma was found. A simple mastoid operation was supplemented with thorough flushing of the wound and abscess cavity with tyrothricin 1:1000 in distilled water. The wound was then lightly packed with gauze saturated with crude penicillin, diluted 1:1 with saline. The skin incision was closed with the exception of the lower half inch, out through which the end of the gauze was brought.

The culture at operation showed a heavy growth of beta hemolytic streptococcus. Culture of a piece of the gauze packing removed from the mastoid cavity at the first dressing four days later showed that beta hemolytic streptococci were still present. The penicillin and tyrothricin applied locally at the operation had not sterillized the wound bacteriologically, but clinically the organisms had lost their virulence, since there was no pus, no swelling, redness, tenderness or other evidence of wound infection. No further packing or irrigation was necessary. If at the first dressing or any subsequent dressing the patient's general condition is good and there is no visual evidence of suppuration, the wound should be inspected, but never touched. There is no better way to infect a clean wound with skin organisms than by probing or inserting packs.

This child would have recovered following the operation if no local chemotherapy had been used, but the immediate bactericidal action of tyrothricin plus the prolonged bacteriostatic effect of penicillin, with which the packing was saturated, so nearly sterilized the wound that the skin incision healed per primam, and so far as dressings or irrigations were concerned the patient was well on the fourth day ofter the operation. Contrast this with the wound left open to granulate and the weeks of dressings that would have been necessary due to wound infection, and you get a clear idea of the value of these drugs in otolaryngology.

SUMMARY

The cardinal principles of successful chemotherapy are: all infecting organisms, both aerobes and anaerobes, must be identified; the bacteria must be sensitive to the drug or combination of drugs

used, and finally the drug must come into direct contact with bacteria in the tissues in an adequate concentration and over a sufficient period of time to allow complete bactericidal or bacteriostatic effect. In acute spreading infections the sulfonamides by mouth or penicillin intravenously or intramuscularly are usually most satisfactory, but in treating chronic mucous membrane or bone infections much depends on the accessibility of the infecting bacteria to the action of the drug. For example, we have often seen pneumococcal meningitis, secondary to chronic ear or frontal sinus infection, temporarily improve following sulfonamides or penicillin given intravenously, intramuscularly or intrathecally, only to recur again and again until the primary focus has been sought out and drained surgically.

The antibacterial substance penicillin is produced by only a few of the many hundreds of strains of the mold Penicillium notatum. Fleming announced the discovery of penicillin in 1929, but more than a decade elapsed before its chemotherapeutic possibilities were fully realized. Since 1940 many reports on methods of preparation, purification and clinical application have been published in England and in this country, but with few exceptions these studies have been directed toward the use of penicillin in the treatment of pneumonia, meningitis, septicemia and other systemic infections. It is not yet possible to make penicillin synthetically and the available supply of the sodium or calcium salt of penicillin for intramuscular or intravenous injection is still very limited; but crude or unrefined penicillin can be made in any bacteriologic laboratory and is often very effective for local application.

Otolaryngologists have an exceptional opportunity for developing the local use of antibiotic substances in the treatment of infection, since most of the organisms in ear and upper air passage infections are gram-positive and sensitive to the action of one or more of these drugs.

Acute infections that are not walled off by the natural defenses of the body and those extending to the meninges or blood stream must be treated by saturating the entire body with antibacterial drugs; but the local use of these substances to aid in the cure of sinus, ear and pharynx infections has been ignored, with the exception of sulfonamide powders, which are usually applied as a routine measure at the conclusion of an operation, with no knowledge of the identity of the infecting bacteria or their sensitivity or resistance to this drug.

The discussion in this paper is limited to: (1) the local use of a solution of sulfadiazine to inhibit the growth of pyogenic organisms in the nose, nasopharynx and pharynx and thus prevent many of the ear and sinus complications of the common cold. (2.) the local use of tyrothricin or crude penicillin in the treatment of acute and chronic infections of the ears and upper air passages. If the conditions of successful chemotherapy mentioned above are fulfilled, it is possible to cure an acute maxillary sinus infection with one irrigation, or so completely to sterilize a wound, that following an extensive surgical operation for acute osteomyelitis of the frontal sinus and skull the incision heals per primam with no redness, swelling, suppuration, or other evidence of wound infection. (3.) the local treatment of hyperplastic lymphoid tissue in the nasopharynx for the prevention or cure of impaired hearing, secondary to partial or complete obstruction of the eustachian tubes, for recurring colds with ear or sinus complications and for some cases of asthmatic bronchitis, especially in children.

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XXIII

MUCOCELE IN FRONTAL AND ETHMOIDAL SINUSES. SIMPLIFIED SURGICAL TREATMENT

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A mucocele may occur in any of the nasal accessory sinuses but is most frequently found in the frontal and anterior ethmoid cells, occasionally in the antrum and rarely in the sphenoid sinus.

Howarth[1] reported five cases with a history of injury in a series of fourteen cases. In one of my cases an external frontal, ethmoid operation had been performed 27 years before. In a second case, an antrum and ethmoid operation had been done 15 years before.

Thomson and Negus[2] state that a mucocele usually occurs with an "obstruction in the outlet of the cavity and it may be caused by blockage and cystic dilatation of a gland." It is reasonable to suppose that such an obstruction to a mucous gland might well occur secondary to an injury or more probably following an obstruction produced by a chronic inflammatory process producing "a cystic dilatation of a mucous gland."

Lobell[3] feels that a degenerative process on a chronic inflammatory basis is very likely the major factor in the development of a mucocele.

Logan-Turner[4] states that the wall of a mucocele consists of epithelium, usually cuboidal in type, with occasional islands of cilia.

The mucocele usually contains a sterile thick gelatinous mucus, often of a yellow brown color, rarely cholesterol. If pus is present the lesion is called a pyrocele.

By a process of slow constant pressure there is a slow absorption of bone, and often the floor of the frontal sinus and the lamina papyracea are destroyed. This destruction advances with the increasing size of the mucocele.

From my cases and those observed in the literature, a slight disturbance of vision progressing to a diplopia and often accompanied

Read before the Sixty-Sixth Annual Meeting of the American Laryngological Association, New York, N. Y., June 7, 1944.

by a dull headache, which may be increased on movement of the eye, constitutes the first symptom of a mucocele. Thus, these patients usually come first to the attention of the ophthalmologist.

The onset is insidious and usually without pain. Three of my patients had had several changes of glasses before the usually globular elastic swelling appeared at the inner angle of the orbit.

In one of my patients, a soldier, severe headaches occurred and in the absence of ocular changes or swelling, malingering was suspected. A density of the frontal sinus, so slight as to be questionable, was the only finding. After some weeks an external exploration revealed a mucocele completely filling a large frontal sinus.

Later the eye is displaced forward, downward and laterally. Diplopia, limited motion of the globe and a variable degree of impaired vision may be present. Ptosis of the lid, epiphora, and even atrophy of the optic nerve may occur.

There may be a "parchment-like" crackling on palpation, and if pulsation is present it means that the posterior wall of the frontal sinus or the floor of the ethmoid, or both, have been absorbed, and the mucocele is lying in contact with the dura mater.

If infection leads to a pyrocele, the swelling may be red and tender, and the temperature may be much like that in an acute frontal sinus infection.

Intranasal examination is usually marked by the absence of polypi or pus. In three of my cases I was impressed with the distinct expansion and lowering of the ethmoid bulla which was smooth.

Early, there are no characteristic roentgenographic findings but with advancement, defects in the orbital and sinus wall occur. The frontal sinus loses its scalloped appearance and the marginal densities become smooth and regular.

In regard to the differential diagnosis osteomas are equally insidious and painless in onset but roentgenograms show a sharp demarcated outline of dense bone, and in advanced cases there is no "parchment crackling" or pulsation on palpation.

Malignant neoplasms are usually much more rapid in growth and in the cases of my experience have shown redundant, soft, free bleeding tissues in the ethmoid area.

Other lesions to be borne in mind are gummas, aneurysms, fibromas, angiomas, lymphoid tumors, meningoceles and chronic sinus infections.

TREATMENT

Chamberlain[5] reported six cases of mucocele of the frontal sinus in 1933. In discussing the paper Dr. D. C. Smith of Boston, remarked, "I have obtained good results by simply leaving the lining of the mucocele in place." This sounded like an excellent idea to me, for a mucocele is lined with epithelium and we were at that time experimenting with skin grafts in an effort to obtain a patent opening into the nose following an external frontal sinus operation. In a mucocele the extension into the anterior ethmoid cells has already been lined by nature with epithelium for us. Why, then, should we destroy it only to have it replaced by contracting, hard, fibrous tissue? Should we remove most carefully only the floor of this extension into the ethmoid area we would have remaining the much sought for epithelial lined duct leading into the frontal sinus. The epithelium lining the mucocele in the frontal sinus can certainly do no harm and pathologically there is no just reason for removing it.

I have used the following plan in operating upon these patients which I believe you will find, if you have not already used it, most satisfactory to the patient and gratifying to the surgeon.

The operation is performed under local anesthesia and a preliminary submucous resection of the nasal septum is done where there is need for better exposure of the middle turbinate.

Should the middle turbinate be wide or cystic in character, it is split with a narrow Bard-Parker knife on a long handle and the lateral one-half is removed with a clean cutting ethmoid forceps. This procedure will give a wide exposure of the ethmoid bulla which the mucocele has usually thinned and displaced downward.

The floor of the ethmoid bulla or anterior ethmoid cell is carefully opened, usually by a single bite or closure of an ethmoid forceps. If the lesion is a mucocele there is a flow of heavy mucoid material. The opening is enlarged by gently inserting a sphenoid biting forceps, removing as much of the floor of the mucocele as possible without injury to its lateral walls which are covered with epithelium and must be preserved.

The thumb is then placed in the upper inner angle of the orbit and the parchment-like bone is pressed back into a normal position. The diplopia is often immediately reduced and improves rapidly thereafter. Novocain may be injected under the skin first, should there be undue pain on pressure.

No drain is placed in the nose. The patient is ready to leave the hospital in one or two days and the only after-treatment is an occasional application of a two or five per cent solution of silver nitrate, should granulations occur.

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XXIV

HISTOLOGIC OTOSCLEROSIS

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This paper reports the incidence of histologic otosclerosis in the laboratory's collection of serially sectioned human temporal bones, describes certain aspects of the lesions found and correlates them with the clinical observations. In most of the patients who had otosclerosis stapedial ankylosis had not occurred and the presence of the disease was not suspected until the sections were examined. The proportion of otosclerotic ears in the collection has therefore not been influenced by conscious selection of such ears for sectioning.

Incidence of Histologic Otosclerosis.—For the statistical part of the study every ear in the collection is used, provided enough sections are available to assure absence of an otosclerotic area. The material (Table 1) that meets this requirement is almost equally divided between Whites and Negroes (585 and 576 patients, respectively). In each racial group about one-third of the patients were females. Sections of both ears are available for 80.3 per cent of the 1161 patients.

TABLE 1.—DISTRIBUTION, BY AGE, RACE AND SEX, OF THE 1161 PATIENTS WHOSE EARS HAVE BEEN HISTOLOGICALLY EXAMINED FOR OTOSCLEROSIS.

Age-Group (Years)	Totals	White Male	White Female	Negro Male	Negro Female
Under 5	161	37	30	48	46
5 to 9	26	8	9	5	4
10 to 14	33	13	6	5	9
15 to 19	55	14	12	16	13
20 to 29	151	35	22	55	39
30 to 39	176	48	40	62	26
40 to 49	210	77	25	86	22
50 to 59	175	67	21	76	11
60 to 69	121	61	21	29	10
70 plus	53	32	7	13	1
Totals	1161	392	193	395	181

From the Otological Research Laboratory, The Johns Hopkins Medical School.

Presented, by invitation, at the meeting of the Otosclerosis Study Group at Chicago, October 11, 1943.

Table 2 gives the distribution, by age, race and sex, of the patients in whose ears otosclerotic areas have been found. Comparison of Tables 1 and 2 shows that only one of the 161 children less than five years old had histologic otosclerosis. This proportion is so low, in comparison to that in any of the older groups, that it is evident otosclerosis seldom begins to develop before the age of five years. The group should therefore be excluded from calculations of the incidence of the disease.

TABLE 2.—DISTRIBUTION, BY AGE, RACE AND SEX, OF THE 49 PATIENTS WITH HISTOLOGIC OTOSCLEROSIS.

Age-Group (Years)	Totals	White Male	White Female	Negro Male	Negro Female
Under 5	1	1	..	..	..
5 to 9	1	..	1	..	..
10 to 14	2	..	2	..	..
15 to 19	2	..	2	..	..
20 to 29	3	2	..	1	..
30 to 39	10	4	5	1	..
40 to 49	17	8	7	1	1
50 to 59	5	3	1	1	..
60 to 69	6	4	2	..	..
70 plus	2	2	..	..	..
Totals	49	24	20	4	1

Table 3 summarizes the data about the incidence of histologic otosclerosis in the patients five years of age or older, grouped by race and sex. For the Whites, the incidence is 1 in 12, for the Negroes 1 in 96. The large racial difference in this material warrants the conclusion that in the general population, also, otosclerosis occurs much more often in Whites than in Negroes. Clinical experience agrees with this histologic observation.

Twenty of the 163 female Whites five years of age or older, and 23 of the 355 male Whites, had histologic otosclerosis. These figures give an incidence of nearly 1 in 8 for the females and 1 in 15 for the males. The number of cases is sufficient to warrant the conclusion that the greater incidence of otosclerosis in the females is not due to chance in this material but is significant for the general population. For the Negroes the number with otosclerosis is too small to warrant conclusions with respect to a sex difference in the incidence.

TABLE 3.—INCIDENCE OF HISTOLOGIC OTOSCLEROSIS, BY RACE AND SEX, IN THE PATIENTS WHO WERE 5 YEARS OF AGE OR OLDER.

Group	Total Number In Group	Number Who Had Otosclerosis	Proportion With Otosclerosis	
			Per Cent	Approximate Rate
White females	163	20	12.3	1 in 8
White males	355	23	6.5	1 in 15
Negro males	347	4	1.1	1 in 87
Negro females	135	1	0.7	1 in 135
All whites	518	43	8.3	1 in 12
All negroes	482	5	1.0	1 in 96

The highest incidence of histologic otosclerosis in this material, for any group of significant size, is in the white women 30 to 49 years old; 12 of the 65, or approximately 1 in each 5½ women of this age-group, had the disease (Tables 1 and 2). For white men the highest incidence is also in the age-group 30 to 49 years; 12 of the 125, or approximately 1 in each 10 had the disease.

In both sexes the incidence is much lower in those who were 50 or older at the time of death than in those who were 30 to 49. The figures for the older groups are: white women, 3 of 49, or about 1 in 16; white men, 9 of 160, or about 1 in 18. The marked drops in the incidence of otosclerosis in the older groups certainly were not caused by the disappearance of otosclerotic areas that had been present at an earlier age, and previously there has been no reason to suspect that the disease may be associated with probable length of life.

The above analyses of the data show the factors of race, sex and age to be so important in the incidence of histologic otosclerosis that the frequency with which it occurs in the entire material in the laboratory merely reflects the relative sizes of the component subgroups. The figure of approximately four per cent, or 1 in 25, would be much larger if the material contained the same proportions of Whites and of females as does the general population of this country.

Location of Otosclerotic Areas.—Data about the 49 patients with histologic otosclerosis are presented in Table 4. For 46 of the patients sections of both ears are available; 32 had bilateral otosclerosis, 14 had unilateral. Nine of the 14 patients with unilateral otosclerosis were white women, 3 were white men. Twenty-two

ears had 2 otosclerotic areas each, and 5 ears had 3 areas each. The total number of otosclerotic areas in the 81 ears is 113.

Otosclerosis was present anterior to the oval window in 65 of the 81 ears. Most of these areas had extensions backward along the superior or the inferior margin, or along both margins, of the oval window. These extensions are not counted as separate areas. Variations in the amount of these extensions will be considered later.

The round window region is next to the oval window region in number of otosclerotic areas. Twenty-five of the 81 ears had an otosclerotic area at some part of the attachment of the round window membrane; usually it was along the lateral part of the attachment, sometimes also anteriorly or posteriorly. Only in 3 of the 25 cases was the area near the round window continuous with the area anterior to the oval window.

The number of otosclerotic areas in each of the other locations in which they occur in this material is:

Stapedial footplate (primary areas only, not including cases of ankylosis)	10
Anterior part of cochlear capsule	6
Inferior part of cochlear capsule (not reaching the internal auditory canal)	5
Anterior-inferior part of cochlear capsule	1
Fundus of internal auditory canal (inferior part)	2
Superior part of cochlear capsule	1
Cochleariform process (limited to this structure only)	1

This material is in agreement with the generally accepted belief that otosclerotic areas occur most often anterior to the oval window, but it does not support the idea that otosclerosis begins in the fissura ante fenestram. The larger otosclerotic areas anterior to the oval window of course include the fissural region, but it is quite impossible in such cases to determine whether or not the pathologic change began in the tissues of the fissura. Positive evidence is afforded, however, by the sections of four ears, each of which has the fissura clearly separate and distinct from a small otosclerotic area near the oval window. Furthermore, each of the 48 otosclerotic areas not anterior to the oval window obviously originated independent of the fissura. The 10 otosclerotic areas which were limited to the stapedial foot-

TABLE 4.—DATA ABOUT THE 49 PATIENTS WITH HISTOLOGICALLY PROVEN OTOSCLEROSIS. COLUMN HEADINGS ARE EXPLAINED IN THE TEXT.

Autopsy Number	Sex, Race, Age	Ear	Areas Anterior to Oval Window: Size of Main Portion	Extensions: Sup.	Extensions: Inf.	Marginal Cartilage Replaced	Stapedial Ankylosis	Active	Areas near Round Window	Active	Areas in Other Locations	Active	HEARING: Audiogram or Conversation	512 Fork: Air: Bone	512 Fork: BC Time	Miscellaneous Observations
10893	F-W-8	R	Large	½	All	Compl., Exten.	No	+	Lat., mod. size	+	No		Good to 16384~	AC>BC	Good	Hearing tested 2 days before death
		L	Mod., deep	½	All	Compl., Exten.	No	+	R. and L., Large	+	No		Good to 16384~	AC>BC	Good	
9705	F-W-17	R	Small, superf.	No	No	No	No	+	No		↑ Ant. to Cochlea & Inf. to Basal Turn	+	Good to 16384~	AC>BC	Good	Positive Family History of Deafness
		L	Small, superf.	No	No	No	No	+	No		No		Good to 16384~, & sl. 2048 dip	AC>BC	Good	
16477	F-W-14	R	Small, deep	No	¼	Partially	No	–	No		No		Good to 4096~, & 8192 at 30 db.	AC>BC	Good	Area surrounds Fissura a.f.
		L	No				No		No		Footplate, ant. pt.	–	Good to 10321~	AC>BC	Good	Only area is limited to Stapes.
4784	M-W-74	R	Mod., deep	½	9/10	Compl., Exten.	No	–	No		Footplate, post. ½	–	Mod. high-tone loss above 2048~	AC>BC	Good	Neither stapedial area is continuous with area outside Ov. W.
		L	Mod., deep	½	½	Complete, Ant.	No	–	No		Footplate, post. ½	–	Like R., except 8192 not heard	AC>BC	Good	
3985	F-W-45	L	Mod., deep	No	1/5	Compl., Ant.	No	+	No		No		Good, at least to 8192~ (2-A audiom.)	AC>BC	Good	Other Ear: No Otosclerosis; good hearing
11282	M-W-42	L	Small, deep	No	¾	Partially	No	–	No		No		Good to 10321~	AC>BC	Good	Other Ear: No Otosc.; Impaired above 1024.
15726	M-W-37	R	Mod., deep	¼	½	Compl., Exten.	No	+	Lateral, Small	–	No		Mod. high-tone loss above 2048~	AC>BC	Good	
		L	Mod., deep	No	½	Compl., Exten.	No	+	Lateral, Large	+	No		Mod. high-tone loss above 2048~	AC>BC	Good	
16257	F-W-36	L	No				No		No		Ant.-Inf. to basal turn	–	Good to 10321~	AC>BC	Good	Other Ear: No Otosclerosis.
11124	M-W-38	R	No				No		No		Inf. wall fundus I.A.C.	+	Good to 10321~, & mod. 4096 dip	AC>BC	Good	In neither ear does otosc. area encroach on nerve or on Sc. tymp.
		L	No				No		No		Inf. wall fundus I.A.C.	+	Good to 8192~, & mod. 4096 dip	AC>BC	Good	
10817	M-W-35	R	Small, superfic.	No	No	Partially	No	–	Lateral, Small	–	No		Mod. high-tone loss above 1024~	Unk.	Unk.	(Unk. = Unknown, or Fork tests were not made.) Probably AC>BC.
		L	Small, superfic.	No	No	Partially	No	–	Lateral, Small	–	No		Mod. high-tone loss above 1024~	Unk.	Unk.	
16352	M-W-64	R	Mod., deep	¼	¼	Compl., Exten.	No	–	No		No		Good to 8192~ (Excellent for Age)	AC>BC	Unk.	
		L	No				No		No		↑ Footplate, post. end & 2. Cochlear Turn is Pos.	–	Slight loss(?) all tones (Ward noisy)	AC>BC	Unk.	
13033	M-W-42	R	Mod., deep	No	1/3	Compl. (1 sec.)	No	+	No		No		Good to 10321~	AC>BC	Good	Other Ear: No Otosc.; Hearing like Right.
15724	F-W-40	R	Mod., deep	No	1/3	Partially	No	–	No		No		Marked h.-t. loss above 1024~ (like L.)	AC>BC	Good	Other Ear: No Otosclerosis.
12411	M-W-42	R	No				No		No		↓ Ant. to Cochlea & Inf. to Cochlea	+	Mod. high-tone loss above 2048~	AC>BC	Good	
		L	Large	¼	¾	Partially	No	+	No		↑ Ant. to Cochlea & Inf. to Cochlea	+	Total Deafness (Acoustic Tumor)	n.h.	n.h.	(n.h. = not heard)
13787	F-W-31	L	Small, deep	No	1/3	Compl., Ant.	No	–	No		No		Total Deafness (Schilder's Dis.)	n.h.	n.h.	Other Ear: No Otosc.; Totally Deaf.
4904	M-W-51	R	Small, deep	No	No	No	No	–	Lateral, Small	–	Ant. to Cochlea	–	Total(?) Dfs. (Masking not used)	Unk.	Unk.	Rt. has Obliter. Labyrinthitis except lower basal turn.
		L	Small, deep	No	No	Partially	No	–	Lat. and P., Mod.	–	Ant. to Cochlea	–	Mod. high-tone loss above 2048~	AC>BC	Good	
4882	M-W-71	R	No				No		Lateral, Small	–	No		Marked h.-t. loss above 2048~	AC>BC	Good	In both ears, Cochlear Atrophy is limited to lower basal turn.
		L	Small, deep	1/5	1/3	Compl., Ant-Inf.	No	–	Lateral, Small	–	No		Marked h.-t. loss above 2048~	AC>BC	Good	
16166	M-W-33	R	Small, deep	No	No	No	No	–	Lat.-Post., Mod.	–	No		Good to 10321~	AC>BC	Good	
		L	Small, deep	No	No	No	No	–	Lateral, Small	–	No		Good to 10321~	AC>BC	Good	
11095	M-W-47	R	No				No		No		Footplate, Exten.	–	Mod. h.-t. loss above 1024~	AC>BC	Unk.	Pt. drowsy; thresholds only approx.
		L	No				No		No		Footplate, Exten.	+	Loss 40-60 db. except 1024 (at 25)	AC>BC	Unk.	Hist. of poor hearing, L., for 12 yrs.
4435	M-W-63	R	Mod., deep	¼	¼	Partially	No	–	Lateral, Mod.	–	No		Gradual drop: 64-20 db.; 4096-85	AC>BC	Short	Both Cochleae have marked degree nerve atrophy in all turns.
		L	No				No		Ant. and L., Large	–	No		Grad. drop: 64-35; 2048-70; 4096 n.h.	AC>BC	Short	
13035	M-W-21	R	Mod., deep	¼	½	Compl., Exten.	No	+	No		No		Slight loss all tones, & 4096 dip	AC>BC	n.h.	Brain Stem glioma may have caused hearing loss, R. ear.
		L	Mod., deep	¼	¼	Partially	No	+	No		No		Good to 10321~	AC>BC	Good	
11173	F-W-34	L	Small, superf.	No	½	No	No	–	No		No		Mod. loss all freq. except 4096, 8192	Unk.	Unk.	Tumor in I.A.C. No otosc. in other ear.
8639	F-W-60	R	Mod., deep	No	½	Compl., Ant.	No	–	No		No		Good to 4096~	AC>BC	Good	Other Ear: No Otosclerosis.

12550	F-W64	R	Mod., deep	1/2	1/4	Compl., Exten.	No	−	No		No		Mod. high-tone loss (Normal for Age)	AC>BC	Short	Other Ear: No Otoscl.; Hearing like Rt.
15758	F-W41	R	No				No		No		Footpl. + Ant. Crus	−	Good to 13004~	AC>BC	Good	
		L	No				No		No		Footpl. + Ant. Crus	−	Good to 13004~	AC>BC	Good	
8633	F-W38	R	Large	No	All	Compl., Ant.	Contact (2sec)	−	No		No		Good to 13004~	AC>BC	Good	Two tests; last one 2 months before death. Occ. tinnitus, mild.
		L	Large	No	All	Compl., Ant.	Contact (?)	+	No		No		Good to 13004~	AC>BC	Good	
15440	F-W45	R	Small, superf.	No	1/3	Partially	No	−	No		No		Good to 10321~	AC>BC	Good	Hearing tested 4 days before death.
		L	Small, superf.	No	No	No	No	−	No		No		Good to 10321~	AC>BC	Good	
10692	M-W47	R	Large	All	1/3	Compl., Exten.	No	−	No		No		Slight loss(?) (Ward noisy)	AC>BC	Good	History, Audiograms, Photo-micrographs in text.
		L	Large	All	1/3	Compl., Exten.	Yes, 3 Regions	+	Ant., Large	+	No		Loss 50-60 db. (last heard - 5793)	BC>AC	Good	
8802	F-W58	R	Large	All	All	Compl., Exten.	Yes, Anterio	+	No		No		Total or Subtotal Deafness (Ac. tumor)	Unk.	Unk.	Hist. of Impaired Hearing for 11 years. Severe tinnitus.
		L	Large	All	All	Compl., Exten.	Yes, Anterior	+	No		No		Loss 50-60 db. all freq. to 10321	BC>AC	Good	
9179	F-W47	R	Huge, contin.	All	All	Compl., Exten.	Yes, Extensive	+	A., L., P., Large	+	Most of Coch. Caps.	+	65-90 db. Loss (last heard - 2896~)	BC>AC	Short	Other Ear: No Sections. (See text)
3934	M-W68	R	Very Large	P. 3/4	All	Compl., Exten.	Yes, Posterior	+	No		No		Extreme Loss (Test Unreliable)	Unk.	Unk.	Cochl. Atrophy is limited to lower basal turn, both ears. (See text)
		L	Large	P. 1/2	All	Compl., Exten.	Yes, Post. part	+	No		No		Like R ear (Explanation in text)	Unk.	Unk.	
8622	F-W44	R	Large	All	All	Compl., Exten.	Yes, 2 Regions	+	No		No		Hysterical (?) Total deafness	n.h.	n.h.	Claimed total deafness for 25 years. Both cochleae essentially normal.
		L	Large	?	?	Unknown	Unknown	+	Unknown		Unknown		Like R. ear (Discussed in text)	n.h.	n.h.	
7722	M-N24	L	Very Large	All	All	Compl., Exten.	Yes, Post. part	+	No		? Ant. to Cochlea & Med. to basal turn	+	No audiogram - Otitis since age 7.	BC>AC	Good	Other Ear: No Otosclerosis.
7772	M-N40	?	Very Large	All	All	Compl., Exten.	Yes, 2 Regions	+	A. and L., Large	+	Inf. to basal turn	+	"Hard of hearing for ten years."	BC>AC	Unk.	Only one ear rec'd; record of side lost.
H315	M-N50	R	No				No		Lateral, Large	+	Superior to Cochlea	+	Subtotal Deafness (Ac. myeloid leuk.)	n.h.	n.h.	Other Ear: No Otoscl.; Hearing like Rt.
10463	M-N39	R	Small, deep	No	No	Partially	No	−	No		No		Hosp. history: "Hearing seems normal"	Unk.	Unk.	
		L	Large	1/3	1/3	Compl., Exten.	No	−	No		No		Like R. (Probably watch + voice used)	Unk.	Unk.	
8970	F-N49	R	Small, deep	No	1/4	Partially	No	−	No		No		Unknown (Pt. comatose on admission and no statement by family)	Unk.	Unk.	Rt. M.T. markedly thickened L.: Ant. sections of area missing.
		L	Unknown	No	1/3	Compl. (sec.)	No	−	No		No			Unk.	Unk.	
9678	M-W1	R	Mod., deep	1/2	All	Partially	No	+	Lateral, Small	+	No		Unknown (Pt. 1 yr., 10 days old)	Unk.	Unk.	Terminal otitis m., bilat.
		L	Mod., deep	All	No	Partially	No	+	Lat. and A., Mod.	+	No			Unk.	Unk.	
10705	M-W45	L	Large	1/2	1/3	Compl., Exten.	Yes, Anterior	+	No		No		"Poor", ? tinnitus, for 3 1/2 wks., Left	Unk.	Unk.	Other Ear: No Otoscl.; chr. pur. otitis m.
11136	F-W18	R	No				No		No		Footplate, Ant. 1/2	+	Hosp. history: "Hearing normal"	Unk.	Unk.	Other Ear: No Otosclerosis.
10975	M-W51	R	Very Large	2/3	1/2	Compl., Ant.-S.	Contact only	+	No		No		Progr. loss for 4 yrs., Rt. first. Pt. thinks R. very poor, L. about 50%.	Unk.	Unk.	Bilateral frontal glioma. Both cochleae essentially normal.
		L	Very Large	1/4	2/3	Compl., Ant.-S.	Contact only	+	No		No			Unk.	Unk.	
10633	M-W68	R	Mod., deep	1/2	1/2	Compl., Exten.	Contact only	−	No		No		Probably good for age; no mention of deafness in hosp. history.	Unk.	Unk.	
		L	Small, deep	1/3	1/3	Compl., Exten.	Contact only	−	No		No			Unk.	Unk.	
11170	M-W28	L	Very Large	1/4	All	Compl., Exten.	Contact (sec.)	+	A. and L., Large	+	No		Partial test: mod. h.-t. loss above 1024	Unk.	Unk.	Areas joined. Other Ear: no sections.
10913	F-W39	L	Small, deep	1/4	1/4	Compl., Ant.	No	+	No		No		Probably good (No mention in hosp. hist.)	Unk.	Unk.	Other Ear: No Otosclerosis.
11093	F-W45	R	No				No		A., L., P.; Large	+	No		Probably good - No mention of hearing, or deafness, in hosp. hist.	Unk.	Unk.	Superficial Exostoses near both oval windows, anterior third.
		L	No				No		A., L., P.; Large	+	No			Unk.	Unk.	
C 1182	F-W10	R	Small, deep	1/2	All	No	No	+	No		No		History: Good hearing, both ears, until acute otitis, R., 3 wks. antemort.	Unk.	Unk.	Suppuration of petrous pyramid, R., & meningitis.
		L	Small, deep	1/2	1/3	No	No	+	No		No			Unk.	Unk.	
3917	M-W48	R	Small, deep	No	1/3	Partially	No	−	No		No		Good for whispered and conversational voice (Refused further tests)	Unk.	Unk.	Above middle third of R. oval window is an independent otoscl. area.
		L	Small, deep	1/3	1/3	Partially	No	−	No		No			Unk.	Unk.	
10882	M-W51	R	Small, deep	No	No	Partially	No	−	Lateral, Small	−	No		Probably good - No complaint about ears, hears conversation well.	Unk.	Unk.	
		L	Small, deep	No	No	Partially	No	−	Lateral, Small	−	No			Unk.	Unk.	
15103	M-W42	R	Mod., deep	1/7	1/7	Compl., Ant.	No	−	No		Footplate, middle 1/3	+	History: Good hearing, both ears, until otitis, Rt., 4 months antemort.	BC|AC	Good	Mastoiditis, suppuration of petrous pyramid, and meningitis.
		L	Small, deep	1/7	1/2	Compl., Exten.	No	+	No		No			AC)BC	Good	

TABLE 5.—SIZE DISTRIBUTION OF OTOSCLEROTIC AREAS ANTERIOR TO OVAL WINDOW, AND SUBDIVISION BASED ON GROWTH ACTIVITY AT TIME OF DEATH.

Size of Area	Totals	Active	Quiescent
Small, deep	19	4	15
Small, superficial	7	2	5
Moderate (all deep)	18	9	9
Large	13	10	3
Very large	6	6	0
Huge	1	1	0
Unknown	1	0	1
Totals	65	32	33

plate not only arose independent of the fissura but also afford evidence against the hypothesis that otosclerosis begins as a metamorphic reconstruction of the bone caused by mechanical stresses and strains transmitted to the otic capsule through the rest of the base of the skull. The presence of the annular ligament makes impossible the transmission of such forces to the stapedial footplate.

In size, the otosclerotic areas of this material vary from ones less than a millimeter in the longer axis to one that had replaced the entire cochlear capsule, except for part of the anterior region and the modiolus, and had extended into the oval and the round window regions.

Activity of the Otosclerotic Areas.—On the basis of structural differences, as deduced from histologic study, several stages of development and metamorphosis of an otosclerotic lesion can be distinguished. Many articles have been published about the appearances an otosclerotic area may have, and controversies have arisen over the exact order of events. The present material contains examples of most of the appearances that have been described. This phase of the subject of histologic otosclerosis, however, will not be discussed or illustrated, because to do so adequately would make this paper unduly long and would require more time and space than its possible contribution to knowledge justifies.

It is sufficient for most purposes to make two broad groups of otosclerotic areas, those which show histologic evidence of recent growth and those which do not. Whether or not an area is growing is a point on which everyone familiar with the histologic appear-

ances of otosclerosis agrees, in spite of differences of opinion in regard to how it grows. The terms "active" and "quiescent" are used to describe these groups. Often one otosclerotic area contains all stages of the process. For purposes of the present report an area is classified as active if any portion appears to have been growing, or undergoing rapid internal changes, at the time of death. Such areas are indicated in Table 4, under the heading Active, by plus (+) symbols; the quiescent areas are indicated by minus (—) symbols.

Active and quiescent otosclerotic areas were present in this material in almost equal numbers, 57 and 56 respectively. Most of the small areas were quiescent, most of the large areas were active. Table 5, in which the areas anterior to the oval window are grouped by size, shows that only 6 of the 26 small areas in this region were active, in contrast to 17 of the 20 large areas. In the round window region (Table 6) 9 of the 10 small areas were quiescent, but only 1 of the 10 large areas was quiescent.

TABLE 6.—SIZE DISTRIBUTION OF OTOSCLEROTIC AREAS IN THE ROUND WINDOW REGION, AND SUBDIVISION BASED ON GROWTH ACTIVITY AT TIME OF DEATH.

Size of Area	Total	Active	Quiescent
Small	10	1	9
Moderate	5	2	3
Large	10	9	1
Totals	25	12	13

For the otosclerotic areas anterior to the oval window, the somewhat arbitrary classification into size-groups was made as follows: An area was classified *large* if it extended from the oval window margin to the endosteum of the basal turn of the cochlea (Fig. 1), *very large* if it reached the middle cochlear turn as well as the basal, *moderate* if it extended halfway or more from the oval window to the basal turn but did not reach the latter, and *small* if it extended less than half the distance from the oval window to the basal turn. Because most of the temporal bones were sectioned in the so-called vertical plane, the classification of an area as moderate or as small is sometimes uncertain. For the round window region the classification as to size of otosclerotic areas (Tables 4 and 6) is wholly arbitrary, being based on the impression gained from looking at the sections while having in mind impressions from other cases.

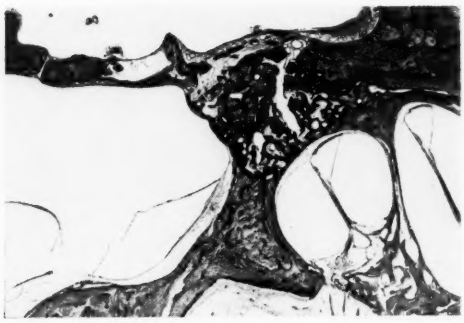

Fig. 1.—An example of an otosclerotic area classified as *large* in Tables 4 and 5. The sizes of areas given other classifications can be visualized from this horizontal section and the text descriptions. Aut. No. 10705, left ear.

All of the 10 areas limited to the stapedial footplate were small, on the basis of sizes in other regions; 3 were active and 7 quiescent. Most of the areas in parts of the cochlear capsule other than near the oval or the round window were large, on the basis of the classification for the oval window region; 12 were active and 4 quiescent.

The relationship that exists between the size of an otosclerotic area and the probability of growth activity at the time of death is a logical one, but the observations do not warrant deductions as to how long any area had been present or as to why growth continued or ceased.

Extent of Otosclerotic Areas Along Oval Window Margins.—For each otosclerotic area anterior to an oval window, the approximate length of its extension backward along the superior and along the inferior margin has been determined by inspection of the serial sections. Table 4, under the heading Extensions, gives these determinations in terms of fractions of the length of the oval window. Only in 7 of the 65 ears was the extent along the superior margin greater than that along the inferior; in the other ears the extensions were either the same or were greater along the inferior margin. In 12 ears the otosclerotic area did not extend back along either margin of the oval window, and in 15 ears it did not involve the superior margin but did extend more or less along the inferior margin. Two ears had otosclerosis along the entire length of the inferior margin of the oval window but none superiorly. The summary of the lengths of the marginal extensions (Table 7) confirms the impres-

sion gained from the larger tabulation; namely, that otosclerotic areas anterior to the oval window usually spread farther back along its lower than along its upper margin. On the basis of the averages, 46 and 30 per cent, respectively, of the marginal lengths, the usual extent of an otosclerotic area along the inferior margin of the oval window is about 50 per cent more than along its superior margin. When consideration is given to the small size of the oval window the difference is not great in absolute dimensions, but it does seem more than can be accounted for by chance alone.

TABLE 7.—DISTRIBUTION OF LENGTHS OF THE EXTENSIONS OF OTOSCLEROTIC AREAS ANTERIOR TO THE OVAL WINDOW ALONG ITS SUPERIOR AND INFERIOR MARGINS, IN TERMS OF FRACTIONS OF THE LENGTH OF THE OVAL WINDOW.*

Margin	None	1/7	1/5	1/4	1/3	1/2	2/3	3/4	9/10	Whole	Average
Superior	27	2	1	9	3	10	1	0	0	9	0.30
Inferior	13	1	1	7	14	9	1	2	1	15	0.46

*Two ears have extensions from below along the posterior part of the superior margin, one has an independent area above the middle third, and for both margins one is unknown because sections are missing.

STAPEDIAL ANKYLOSIS

The stapediovestibular articulation had become ankylosed in only 10 of the 81 ears that had histologic otosclerosis. This number includes all cases with an osseous connection, no matter how small, between the stapes and the oval window margin. The ratio of approximately 1 in 8 increases to 1 in 6½, or about 15 per cent, when only the 65 ears with otosclerosis in the so-called region of predilection, anterior to the oval window, are considered.

Six of the ears with stapedial ankylosis are from males and 4 are from females. The number of cases is too small to warrant any conclusions as to a sex difference in the probability of ankylosis in patients who have histologic otosclerosis.

In none of the 10 ears was the entire stapediovestibular articulation obliterated. Usually less than half of the circumference of the oval window was connected to the stapedial footplate by osseous tissue. The sections show more than one osseous connection in some of the ankylosed stapediovestibular articulations, and portions of each annular ligament are normal in appearance

Stapedial ankylosis had not occurred in 55 of the 65 ears with otosclerotic areas in the oval window region. In most of these 55 ears, however, the distance between the otosclerotic area and the

stapes was less than 1/125th of an inch, or 0.2 mm. The marginal cartilage was completely replaced with otosclerotic tissue at some part of the circumference of the oval window in at least 27 of the ears in which ankylosis had not occurred. In 7 of these ears the annular ligament also had been replaced by otosclerotic tissue to such an extent that in one or more of the sections the otosclerotic area appeared to be in actual contact with the cartilage of the stapedial footplate. In the other 20 ears the areas were separated from the stapedial footplate only by the annular ligament. The width of the normal annular ligament (average of 16 measurements, 4 in the center of each quadrant—anterior, superior, posterior and inferior) is 63 micra (0.063 mm., or about 1/400th of an inch). In other words, these 20 otosclerotic areas were separated from the stapes by a distance about equal to one-sixth of the width of the vertical part of the letter "l" of the type most used in printing this journal. The marginal cartilage of the oval window was partially replaced by otosclerotic tissue in 18 ears. Since the marginal cartilage has an average thickness of about 60 micra, most of these 18 otosclerotic areas were less than 0.1 mm. from the stapes. Only in 9 of the 65 ears with otosclerotic areas anterior to the oval window was the marginal cartilage intact.

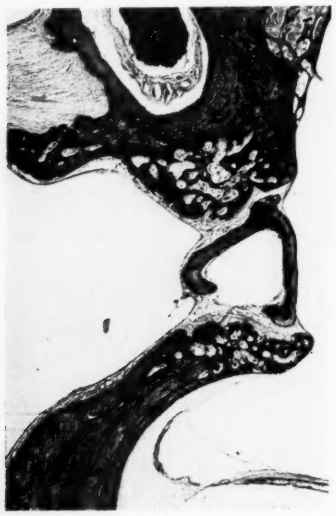

Fig. 2.—Photomicrograph of section through anterior part of the oval window of an ear with active otosclerosis but no loss of hearing and no bony connection to the stapedial footplate in spite of complete replacement, in this region, of the cartilage of the oval window margin. Aut. No. 10893, right ear.

The observations show clearly that most otosclerotic areas in the oval window region grow to within a very short distance of the stapes but do not form osseous connections to it and do not ankylose the stapediovestibular articulation.

HEARING

Hearing tests, including audiograms, were made of 55 of the 81 ears in which histologic examination later revealed otosclerotic

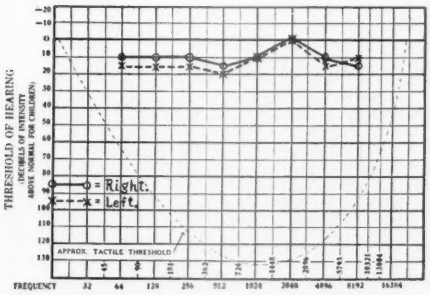

Fig. 3.—The audiogram of a 45-year-old woman with unilateral otosclerosis, histologically demonstrated (see Fig. 4). The thresholds for both ears are within normal range for the conditions under which she was examined.

areas. Some information with respect to hearing is available for 22 of the other ears (see Table 4). Nothing is known about the hearing of 2 patients (4 ears); one was comatose throughout her hospitalization, the other was one year old.

Without exception, the material on which this report is based is in agreement with the opinion that otosclerosis does not cause an impairment of hearing until, or unless, a bony connection becomes formed between some part of the oval window margin and the stapedial footplate. The following two cases well illustrate how good the hearing may be in spite of otosclerotic lesions of the oval window region:

An 8-year-old girl had excellent hearing with both ears for all frequencies, including 16384 cycles, two days before death, which occurred after an operation for a brain stem tumor. The sections show that she had active otosclerotic areas, bilaterally, in the oval and in the round window regions. The articular cartilage of part of each oval window margin had been completely replaced (Fig. 2), but neither stapes was ankylosed.

The audiograms of the second patient are shown in Fig. 3. The thresholds for both ears were nearly the same and were within the normal range for tests in a hospital ward with the audiometer used. Nothing about the examinations caused anyone to suspect that this 45-year-old white woman had otosclerosis. Histologic study of her temporal bones, however, revealed unilateral otosclerosis. Her left ear had an active otosclerotic area anterior to the oval window (Fig.

4) which had completely replaced the articular cartilage of the anterior margin of the oval window and had extended backward about one-fifth of the length of the inferior margin. There was no bony connection between the oval window margin and the stapes.

When lesions such as the two illustrated above do not mechanically interfere with the transmission of sound waves, otosclerotic areas more distant from the stapedial footplate certainly do not do so. Such evidence, of course, does not rule out the possibility, often expounded as an explanation for clinical observations, that otosclerotic lesions frequently cause a degeneration of the cochlear nerve with a resultant severe impairment of hearing of a mixed or of a pure nerve-deafness type. The present material does not support this theory, either.

Four of the ears with histologic otosclerosis are known to have had good hearing for all frequencies up to 16384 cycles, 4 others had good hearing up to 13004 cycles, and at least 10 other ears had good hearing up to 10321 cycles. Thirteen of the 55 ears tested with an audiometer had an impairment of hearing for the frequencies above 1024 or 2048 cycles, but good hearing for all the lower frequencies. Similar impairments of hearing for high tones are of frequent occurrence in patients without histologic otosclerosis, and in them the losses are universally regarded as caused by aging, tubal occlusion, acoustic trauma or some other condition not related to otosclerosis. There is no evidence to indicate that any of the losses limited to high tones in the patients with histologic otosclerosis were caused by the osseous lesion present.

Twenty-three ears had impaired hearing for conversation; 16 of them were tested with an audiometer. Nine of the 23 ears had a total or a subtotal deafness and are therefore of especial interest with respect to the question of cochlear nerve degeneration in otosclerosis. Stapedial ankylosis had occurred in 4 of the totally or subtotally deaf ears, and in 4 it had not occurred. The condition of 1 ear with respect to ankylosis is unknown because the critical sections were lost during preparation. For each of the 4 cases without stapedial ankylosis an obvious cause of the deafness was found: one patient had an acoustic tumor, one had Schilder's disease (encephalitis periaxialis) that had spread to the auditory pathways of the brain stem two months before causing death, one had an obliterative labyrinthitis (etiology and duration unknown), and one had an acute myeloblastic leukemia with an extreme degree of myeloblastic infiltration of both the middle and the inner ear.

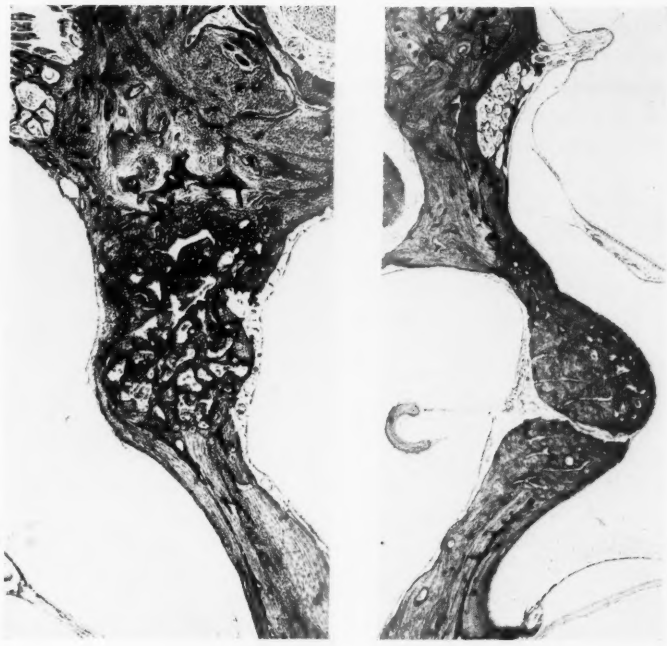

Fig. 4.—This photomicrograph shows the area of actively growing otosclerosis immediately anterior to the oval window of the left ear of the woman whose audiograms are shown in Fig. 3. Her other ear had no otosclerotic area. Aut. No. 3985.

Fig. 5.—Ankylosis, superiorly, of a stapes in a quiescent otosclerotic area. Although the inferior margin of this oval window, including the marginal cartilage, has been replaced by otosclerotic bone, there is no bony connection to the footplate inferiorly in this section. Aut. No. 3934, right ear.

For the 4 ears that had stapedial ankylosis and were totally or subtotally deaf there are also factors that complicate the interpretation of the effect of the otosclerosis on hearing. One patient had an acoustic tumor. Two ears, and they have the most nearly complete ankylosis of any of the material (Fig. 5), are from an extremely emaciated 68-year-old white man who died the day after the attempt to test his hearing. The cause of death was not established, but a contributing factor was chronic bronchitis aggravated by acute starvation; he claimed he had had no food for five days before admission to the hospital. He was unable to cooperate much in the hearing tests and it is unreasonable to believe his deafness was as extreme as it appeared to be, especially when consideration is given to the fact that both cochleae were normal histologically except for moderate atrophy in the lower part of the basal turn. Such atrophy is often present in patients of his age. The fourth ear with stapedial ankylosis and total deafness and the deaf ear for which the critical sections with respect to ankylosis were lost are from a 44-year-old white woman who had been a psychiatric patient for 25 years. She belonged to a wealthy family and claimed to have been totally deaf since the age of 19 years, when she thought her personal maid tried to poison her. She gave no response to auditory stimuli of any kind. Both cochleae were essentially normal, histologically, and the stapedial ankylosis was of only moderate extent in the ear for which all sections are available. The histologic observations are in agreement with the clinical diagnosis, by eminent psychiatrists, that her total deafness was mostly on an hysterical basis. The facts about these cases show clearly that none of the 9 ears which had the most severe hearing impairments of any in this material can be used to support an opinion or argument in favor of cochlear nerve degeneration secondary to otosclerosis as the cause of the extreme deafness.

Three other ears are also of special interest with respect to the question of cochlear nerve degeneration secondary to otosclerosis; two actually had extensive nerve degeneration, the other had the largest otosclerotic area in this material. The two ears are from a 63-year-old white man. Neither stapes was ankylosed, but both ears had a marked degree of atrophy of the cochlear nerve in all turns. The patient had, bilaterally, a moderate to marked impairment of the thresholds for low tones and an extreme loss for high tones; with one ear 2896 cycles was the highest frequency heard, with the other ear 4096 was the highest. With both ears he heard the 512 d.v. steel tuning fork better by air than by bone conduction, and bone conduction time was very short. The hearing tests are typical of those of patients with extensive cochlear nerve atrophy but without histo-

logic otosclerosis, of which the laboratory collection contains several examples. The presence of otosclerosis in the ears of this patient is therefore regarded as only a coincidence.

The ear with the largest otosclerotic area is from a white woman, 47 years old, who had a positive family history of deafness. She had noticed impairment of hearing in this ear about 12 years previously (at the age of 35), and said it had progressed rapidly and that she had tinnitus. The tympanic membrane was normal. The audiogram showed a severe degree of hearing impairment; the thresholds ranged from 65 decibels loss for 64 and 128 cycles down to 90 decibels for 2896, which was the highest frequency she heard. The 512 d.v. steel tuning fork was heard better by bone than by air conduction, but bone conduction time was markedly shortened. Histologically, the middle ear and the cochlea were essentially normal with the exception of an otosclerotic area that had replaced all of the cochlear capsule, except for part of the anterior region and the modiolus, and had extended into the oval and the round window regions. Ankylosis of the stapes had occurred; the footplate was attached to the oval window margin in several short regions rather than continuously. The patient died four days after the hearing tests were made, following an operation to remove a pituitary tumor. The evidence from other patients with pituitary tumors, whose hearing has been tested in the laboratory, makes it highly improbable that the intracranial lesion affected this woman's hearing. The clinical findings with respect to the ear of this patient are typical of those usually interpreted, here as well as elsewhere, as indicative of otosclerosis with secondary nerve degeneration. The cochlear nerve and its end-organ are, however, normal histologically. The observations can be interpreted to support the hypothesis that the otosclerotic area impaired the hearing by a toxic action on the normal nerve or end-organ, but it is at least equally plausible that some unrecognized peculiarity of the conductive lesion (stapedial ankylosis) caused more impairment for high than for low tones and also impaired the transmission of bone-conducted sound waves to the cochlear fluids. The other temporal bone of this patient was ruined during preparation, probably it also was otosclerotic.

Otitis media was obviously the cause of the impaired hearing of 2 ears, neither of which had stapedial ankylosis. A metastatic tumor in the internal auditory canal offers a good explanation of the moderately impaired hearing of another ear in which the stapes was not ankylosed. No explanation of the hearing loss is apparent in the sections of the ears of a 51-year-old physician who complained

of a progressive impairment of hearing for four years and who had bilateral otosclerosis without stapedial ankylosis. Hearing tests were not made, so the amount and type of his deafness are unknown. His ears are the only ones in the entire material that might be used to support the view that otosclerosis impairs the hearing before stapedial ankylosis occurs. The support they offer is rendered negligible by the fact that the patient had a glioma of both frontal lobes that had extended backward into other parts of the brain also.

The remaining 5 ears, of the 23 with histologic otosclerosis and impaired hearing for conversation, had stapedial ankylosis without complicating factors, and are the only ones, besides the ear with the largest area, for which a clinical diagnosis of otosclerosis could have been made. The lesion in the left ear of the following case is typical of that present in clinically recognizable otosclerosis:

The patient, a 47-year-old white man, said that his hearing had been poor for four years and was gradually getting worse, that he had continual tinnitus, and that his mother was hard of hearing in one ear. His wife thought he had had impaired hearing in one ear for at least 20 years. The audiograms are shown in Fig. 6. With the left ear, to which he lateralized the sound, the 512 d.v. steel tuning fork was heard better by bone than by air conduction. With the right ear the fork was heard better by air than by bone conduction. Bone conduction time, for the 512 d.v. fork, was normal for each ear with the other masked. The patient denied having had otitis media and the otoscopic examination was negative.

The serial sections of the temporal bones, which were secured at autopsy two hours after death from chronic endocarditis, show essentially normal middle and inner ears except for bilateral otosclerosis. Both ears have an otosclerotic area in the oval window region; the left ear also has an area near the round window. The stapediovestibular articulation of the left ear was ankylosed (Fig. 7.) The otosclerotic area had formed three osseous connections to the stapedial footplate, which were separated from each other by nonankylosed portions of the articulation. The largest osseous connection joined the posterior third of the footplate, both superiorly and inferiorly, to the oval window margin (Fig. 7, *C*); the smaller connections were anterior and near the middle of the superior margin. The otosclerotic area appears to have been actively growing.

In the right ear, for which the Rinne test was positive, the stapes was not ankylosed (Fig. 8), and the otosclerotic area shows no evidence of recent growth. The histologic findings do not ex-

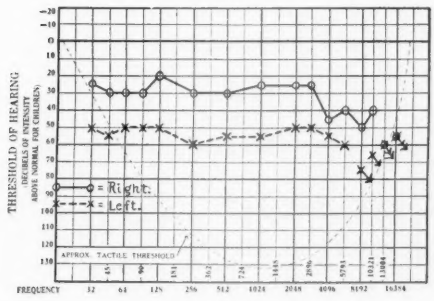

Fig. 6.—The audiograms of a 47-year-old man with bilateral otosclerosis, histologically demonstrated to have unilateral ankylosis of the stapes (Figs. 7 and 8). Other data as to hearing are given in the text.

plain the slight impairment indicated by the audiogram (Fig. 6), and it should be noted that with this ear he heard the whispered voice from a distance of 9 feet and low conversational voice from more than 15 feet. It is therefore probable that part of the impairment indicated by the audiogram was due to the masking effect of room noise (he was examined in a ward), to inattention or to general fatigue from his serious illness. The evidence from other cases in this material, which have been presented above, shows that the mere presence of an otosclerotic area does not cause impaired hearing.

SUMMARY AND CONCLUSIONS

This paper reports the incidence of histologic otosclerosis in the laboratory's collection of serially sectioned human temporal bones, describes certain aspects of the lesions found and correlates them with the clinical observations. The more important statistical facts are:

Forty-nine of 1161 patients had histologic otosclerosis. Sections of both ears are available for 46 of the 49 otosclerotic patients; the disease was bilateral in 32 and unilateral in 14.

The incidence is lowest in the children less than 5 years of age; only 1 of the 161 had histologic otosclerosis.

The highest incidence is in the white women 30 to 49 years old at the time of death; 12 of the 65, or about 1 in each 5½, had histologic otosclerosis.

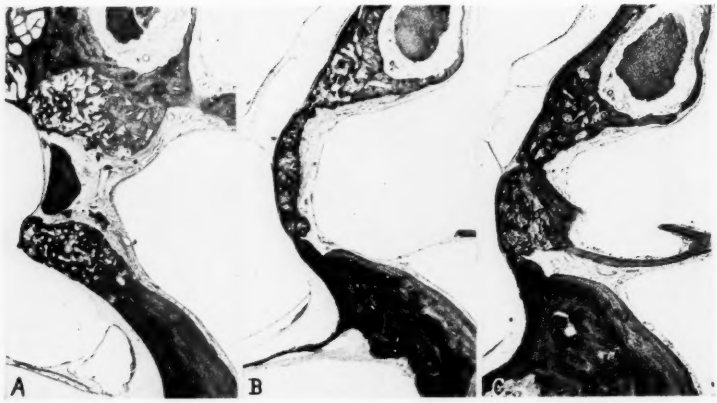

Fig. 7.—*A*, *B* and *C* are, respectively, photomicrographs of vertical sections through the anterior, middle and posterior thirds of the oval window of the left ear of the man whose audiograms are shown in Fig. 6. This otosclerotic area was actively growing and had formed osseous connections with the stapedial footplate in three regions, two of which are shown. See Text. Aut. No. 10692.

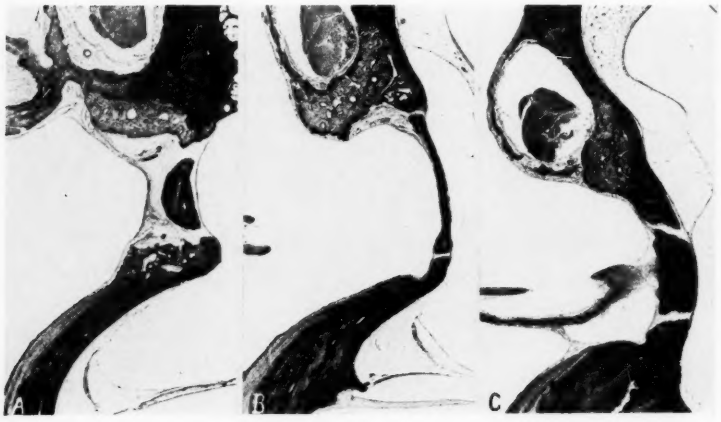

Fig. 8.—*A*, *B* and *C* show the oval window of the right ear of the man whose audiograms are presented in Fig. 6 and are from regions that correspond to those of the left ear shown in Fig. 7. This otosclerotic area is quiescent and there is no ankylosis. See text.

For the patients 5 years of age or older, grouped by race and sex, the incidence of histologic otosclerosis is:

White females:	20	of	163,	or	about	1	in 8.
White males:	23	of	355,	or	about	1	in 15.
Negro males:	4	of	347,	or	about	1	in 87.
Negro females:	1	of	135.				

The 81 ears had a total of 113 otosclerotic areas, of which 65 were located anterior to the oval window and 25 were near the round window. Ten of the areas were limited to the footplate of the stapes and appear to have originated in that structure.

Stapedial ankylosis had occurred in only 10 of the 81 otosclerotic ears. In 45 ears an otosclerotic area was less than 0.1 mm. from the stapedial footplate, but ankylosis had not occurred.

Hearing tests, including audiograms, had been made of 55 of the 81 ears with otosclerotic areas, and general information as to hearing is known for all but 4 of the other ears. Four ears had good hearing for all frequencies up to 16384 cycles per second, 4 had good hearing up to 13004 cycles and at least 10 others had good hearing up to 10321 cycles. Fifteen ears had impaired hearing for the frequencies above 1024 or 2048 cycles and good hearing for the lower frequencies. Twenty-three ears, including the 10 with stapedial ankylosis, had impaired hearing for conversation, but in none of the 13 without ankylosis can the impairment be attributed to the presence of an otosclerotic area. Other conditions, described in the text, had caused total or subtotal deafness in 4 of the ears with stapedial ankylosis, so that for only 6 of the 81 ears with otosclerotic areas would it have been possible to make a clinical diagnosis of otosclerosis.

The above facts, together with others reported in the body of the paper, warrant the following conclusions:

1. A clear distinction must be made, in all discussions of otosclerosis, between clinical otosclerosis and histologic otosclerosis. Failure to keep this distinction in mind can lead only to confusion of ideas.

2. Otosclerosis is often asymptomatic. The disease does not cause impaired hearing unless, or until, ankylosis of the stapediovestibular articulation occurs and prevents normal movements of the footplate of the stapes. Most otosclerotic areas do not cause ankylosis

of the stapes, even when they are located at the anterior margin of the oval window.

3. Atrophy of cochlear nerve fibers or of the organ of Corti does not occur more often in ears with otosclerotic areas than in ears free from otosclerosis. When atrophy does occur in otosclerotic ears it is usually limited to the basal turn and differs in no way from the nerve and end-organ atrophy that is often seen in the sections of ears without otosclerosis. The etiology of cochlear atrophy in ears without otosclerosis cannot, in most cases, be established, and it therefore does not seem logical to attribute to otosclerosis all the cochlear atrophy found in otosclerotic ears.

4. Otosclerosis is primarily a disease of bone, and stapedial ankylosis is but an incidental event that occurs occasionally. No reason is known why one otosclerotic area stops short of ankylosis of the stapes and another area, similar in appearance in every other respect, spreads across the annular ligament to the stapedial footplate.

5. Because the presence of otosclerosis in an individual becomes apparent clinically only when ankylosis does occur, the otologist should attach but little diagnostic importance to a negative family history of deafness, even when well authenticated. He should keep in mind the fact that persons with normal hearing may have otosclerotic areas, that the distance of many such areas from the stapes is less than 0.1 mm., and that growth of the area across the narrow gap would cause deafness.

6. The facts observed in the present material render invalid all conclusions with respect to dominant and recessive genes, that have been drawn from clinical studies of the inheritance of otosclerosis. To obtain a sufficient basis for a reliable determination of the hereditary factors in otosclerosis it would be necessary to examine histologically the temporal bones of all members of many family groups, irrespective of their acuity of hearing. Only thus can the presence or absence of the primary disease, histologic otosclerosis, be determined. Contemplation of the histologic facts reported in this paper leads also to the conclusion that it would be futile to try to eliminate otosclerosis by eugenic measures.

7. Unilateral otosclerosis does occur, and not infrequently.

8. Otosclerosis occurs much more frequently in Whites than in Negroes, and in Whites its incidence is higher in women than in men.

JOHNS HOPKINS HOSPITAL.

XXV

VITAMINS IN OTOLARYNGOLOGY

H. B. PERLMAN, M.D.

CHICAGO, ILL.

In the past decade there has been a rapid growth of interest in vitamins. This interest has not been confined to the laboratory but has rapidly spread to the medical and lay public. A great deal of specially prepared vitamins are now consumed not only under the direction of a physician but more often on the advice of the corner druggist or as the result of direct advertising. This interest in vitamins is shared by the otolaryngologist and is perhaps a natural development of laboratory research in this field. Constant search for a better understanding of many of the obscure diseases that the otolaryngologist is called upon to treat, together with a desire to follow any suggestive lead in therapy and the constant pressure of the patient for help in relieving his illness often spurred on by unwarranted claims of advertisements and inaccurate information, has resulted in a widespread interest and use of vitamins in the practice of otolarygology as in many other fields of medical practice.

It appears that a review of the subject at this time is indicated in an attempt to evaluate present scientific information about the vitamins as it may relate to otolaryngology. In looking for this relationship between vitamin physiology and clinical otolaryngology, the soundest approach is to review the clinical picture of known deficiency states in man and not in the experimental animal. These vitamin deficiency diseases have occurred in outbreaks in various parts of the world, are endemic in some places, and have been reproduced in man under controlled laboratory conditions. It is safe to assume that if otolaryngological manifestations are not found in the full-blown deficiency state, they are not likely to be found in any so-called subclinical deficiency state. On the other hand, if we have otolaryngological signs in the classical deficiency states, we may be permitted the view that the presence of these signs help to define the

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Work done in part under a grant from the Douglas Smith Foundation of the University of Chicago.

Read as a candidate's thesis before the Chicago Laryngological and Otological Society, November 1, 1943.

deficiency state and when correlated with other objective evidence of the specific deficiency, these signs can be of definite importance in clinical otolaryngology. While many vitamin fractions have been identified by controlled animal experiments only a few of the vitamins have been connected with known clinical states in man. These may now be reviewed briefly.

Vitamin A deficiency produces xerophthalmia and night blindness in man. This deficiency state is endemic in some portions of the Orient. A particularly large incidence has been found in some parts of India and in the Malay peninsula. Sexually mature persons with xerophthalmia may have cutaneous lesions of a papular nature due to hyperkeratosis of the hair follicles. As many as five per cent of all hospital admissions in some parts of Java are patients with xerophthalmia[1]. Many cases among children are seen in Latin American hospitals. Furthermore an epidemic of xerophthalmia broke out in Denmark shortly after the last World War.[2] Occasional postmortem examinations have been made on infants and children dying with this deficiency state. In reviewing the clinical picture from reports of these cases and the postmortem findings there is very little evidence of ear, nose or throat pathology. An occasional report of metaplasia of the bronchial and sinus mucous membrane appears in the autopsy findings.[3]

Vitamin B_1 or thiamine deficiency produces a disease called beriberi. This is endemic in certain portions of the Orient as the result of eating a diet largely confined to polished rice. The clinical picture is that of multiple neuritis, not an isolated neuritis, and this neuritis regularly affects the nerves of the legs first—leading soon to muscular and skin atrophy. This is called the "dry type." A "wet type" is characterized by edema and cardiac changes that may cause death. Again in the clinical and pathological reports of these cases otolaryngological signs are singularly scarce. Furthermore this vitamin may be synthesized in the human intestinal tract.[23]

Nicotinic acid deficiency is endemic in some of the southern areas of the United States in the form of pellagra. Ulcerations in the mouth and glossitis are the principal otolaryngological signs. A dry esophagitis with ulcers was also present in a number of the pellagra patients according to Fisher.[22] Dermatitis, dementia and diarrhea are the chief late signs of this disease. Often associated with this deficiency is riboflavin deficiency, another vitamin B fraction. Inflammation of the lips and ragades about the corner of the mouth and nose appear to be its principal clinical signs. Before these symptoms appear, burning of the mouth, tongue and eyes have been noted in carefully

studied groups of patients.[4] During the Spanish Civil War many cases of glossitis were seen. These appeared to respond to vitamin B complex in the form of yeast, better than to nicotinic acid.[5] The ulcers of the tongue and the oral cavity seen in nicotinic acid deficiency tend to become infected with Vincent's organisms and respond to nicotinic acid therapy.[6] Similar chronic ulcers encountered in the clinic, even when no other signs of deficiency exist have occasionally responded to nicotinic acid therapy.[7] Other fractions of vitamin B have been identified only on experimental animals and have no clinical counterpart.

Vitamin C or ascorbic acid deficiency is seen among large numbers of the South African natives beginning work in the mines[8] and has been described years ago in epidemics among sailors long deprived of fresh fruits and vegetables. It has been reproduced in man under controlled conditions.[9] This latter study revealed that an ascorbic acid level of zero in the plasma appeared after 41 days and in the white cell platelet layer after 87 days of complete depletion. No symptoms other than loss of weight appeared for four months. This is corroborated by observations in this clinic that some individuals may have no ascorbid acid in their plasma and yet show no effects of this. After about 132 days of complete depletion there appeared a papular skin eruption and only after about 161 days did the first classical sign of perifollicular hemorrhage develop. An experimental wound now showed delayed healing due to the failure of intercellular substance to form, while after three months of complete depletion wound healing was normal. Capillary fragility tests were normal. No other signs appeared in this controlled depletion experiment. Low ascorbic acid has been found in a few cases of gingivitis at the dental clinic, one or two of the patients having irritations from dentures. These have responded to ascorbic acid therapy.

Vitamin D deficiency produces symptoms during the growth period and these are related to imperfect and delayed bone formation manifested in the clinical picture of rickets. Again no otolaryngological signs are common to this deficiency state. No other known clinical vitamin deficiency states are known although the tocopherols of vitamin E appear to be concerned with the reproductive and nervous functions in animals and vitamin K is important in the formation of prothrombin. The latter information has been used clinically in controlling surgical bleeding of patients with obstructive jaundice and is useful in hemorrhagic states of the newborn before the bacterial flora of the intestinal tract becomes established. With

the aid of bile salts, bacteria in the intestine can apparently mobilize this vitamin.

Criteria for defining known deficiency states in man are still in the process of formulation.[10] They include quantitative chemical determinations. Until these criteria are established the subclinical vitamin deficiency state cannot be seriously considered. In the otolaryngologic literature a number of articles have appeared in which the author has attempted a correlation between vitamins and the diseased states.[11-14]. While a great deal of work was done in attempting to evaluate the general status of the patients studied, the evidence for a vitamin deficiency state was not convincing and the multiplicity of therapeutic procedures with numerous vitamins and endocrines often made it impossible to analyze the results. Furthermore the results reported were not conclusive, but rather could be considered to be within the limits of experimental error, as in the case of repeated audiograms, or could be explained on the kind of cases chosen for therapy—cases in which variations in symptoms could reasonably be expected—such as patients with tinnitus, allergy or Ménière's disease. Generally there appears to be very little basic information in the clinical vitamin studies reported thus far by otolarygologists. This is no reflection on the clinician's work except that the evidence presented in this work did not warrant his conclusion of a definite relationship existing in humans between various clinical pathologic states and vitamins.

In contrast to many necessarily poorly controlled clinical studies the controlled animal experiment and the biochemical studies continue to bring new light on the physiology of the vitamins and suggest possible ultimate application to otolaryngology. However, extreme caution should be exercised in transposing the results of vitamin experiments on animals to the clinic. The following personal observations exemplify this:

From the otologist's standpoint perhaps one of the most suggestive experiments is that of Mellanby on vitamin A depletion of dogs.[15] I studied this problem in the rabbit and confirmed his observations that you can produce marked hyperplasia of the bone at the internal meatus.[16] His preparations showed also extensive degeneration of the spiral ganglion and end organ. That these changes were secondary to pressure effects of the bone narrowing the internal acoustic meatus and not a primary neural effect of the vitamin A depletion is shown by the absence of these findings in the depleted rabbits. In our animals with one exception the amount of new bone

formation was not sufficient to destroy the auditory nerve directly or by interference with the blood supply.

Furthermore, this was verified by testing the cochlear function before and after depletion using the acoustic middle ear muscle reflex as an index of cochlear function. The experiments were carefully controlled and the vitamin A level of the plasma was determined by the antimony trichloride reaction with the aid of a photoelectric colorimeter. The striking pathology of the temporal bones of these vitamin A depleted rabbits as well as those of Mellanby's dogs would at first glance suggest a really important translation value to clinical otology. However, a careful analysis of all the factors concerned does not bear this out. First of all this experimentally produced temporal bone pathology practically has no counterpart in human temporal bone pathology. Of the thousands of human temporal bones that have been studied, there exist but one or two descriptions that resemble this experimental lesion.[17] Furthermore the bony hyperplasia in the experimentally depleted animal is not confined to the internal auditory meatus but rather generally involves the posterior fossa surface as well as the spinal canal. While Mellanby previously thought that the multiple peripheral spinal nerve degeneration he found in his depleted animals was a primary effect, he has now found the explanation in the bony hyperplasia about the spinal nerve foramina.[18]

Similar experiments carried out on adult animals are entirely negative, probably because the reserve of vitamin A stored in the liver is practically impossible of exhaustion. Only growing young animals will show changes. Further these animals even on complete depletion have to be carried for a long period relative to their life span before signs of depletion develop. By this time they are markedly retarded in growth, have xerophthalmia and are generally in such a poor state of health that death may soon intervene.

Another point is brought out in considering the results of our experiments. Other workers reporting on changes in vitamin A depleted rats noted a high incidence of middle ear infections.[19, 20] Our animals did not show this. This again illustrates the danger of transposing results from one experimental animal to another and particularly from the experimental animal to man.

Referring again to clinical or pathological studies of vitamin A depletion in man we find no mention of this temporal bone or cranial base pathology or evidence of deafness. Lastly we are confronted with the fact that vitamin A deficiency as manifested by

its classical signs in man is practically unknown in this country. The adequate vitamin A intake of most of our population plus the fact that liver stores seem adequate through very long periods of depletion explain this. The biophotometer tests for dark adaptation are not considered reliable enough to be proof by themselves of a definite vitamin A deficiency state or at best suggest the extremely mild deficiency state. Reviewing our experimental material in this light, we see that otologic problems in the clinic are not likely to be explained on the basis of vitamin A deficiency. Many of the experimental observations of vitamin depletion in animals have been reviewed from an otolaryngological angle[21] but they should be examined critically along the lines indicated by the analysis of the results of this vitamin A depletion study when considering their clinical application.

The physiology of overdoses of vitamins is still very obscure and as yet offers little prospect of special value to the otolaryngologist. Usually the amount of vitamin in excess of the body need is eliminated. The use of vitamins for a transitory pharmacologic effect— as, for example, producing vasodilation with nicotinic acid — may be mentioned only to point out that it is not directed towards correcting a specific deficiency state. One cannot expect to relieve a long-standing pathological process by inducing a non-specific pharmacologic effect lasting only a few hours. Only changes in transient symptoms may be expected by such treatments.

While a critical analysis of our present knowledge of the vitamins gives us little of proven value as regards the clinical problems encountered by the otolaryngologist, one cannot underestimate the contribution of the laboratory worker, the biochemist and the clinician in increasing our knowledge of basic biologic phenomena of cellular physiology as related to nutrition. Continued growth of our knowledge in this field, together with carefully controlled studies in man, give promise that answers to some of the unsolved problems in otolaryngology may be forthcoming.

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XXVI

TRAUMATIC DEFORMITIES OF THE NASAL SEPTUM

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For a number of years following the early development of the submucous resection of the septum by Killian, Ballenger, Freer and others, rhinologists generally have felt that the technique was adequate. In recent years, however, unsatisfactory results in certain types of cases have led to at least a partial revision of our ideas on the subject and to a number of modified procedures. The object of these newer procedures is not only to relieve the patient of his nasal obstruction, but at the same time to prevent distortion or collapse of the nasal tip and to correct such concomitant deformities as may be in intimate association with the deviation.

A careful analysis of a large series of cases has convinced me that a great many of the cases we see are the result of trauma, recent or ancient, (generally the latter) and, therefore, if we are to restore the nose to its normal status, we must consider first the primary case of injuries to the nose as a prophylactic measure, and second the actual treatment of such deformities where primary treatment has been inadequate.

Trauma to the nose is exceedingly common for obvious reasons, but unfortunately unless it is severe, it frequently is overlooked or neglected. This is particularly true in children who sustain innumerable falls when learning to walk and later at play. Even minor trauma at this stage may have serious consequences because of the immature state of the constituent nasal framework, since ossification is not complete until after puberty. The nasal bridge and the septum, being partly cartilaginous, fail to fracture completely. Instead they may bend or curve slightly as a result of injury and continue then to develop into a deformity which becomes fully manifest only in later years. A child whose nose has been injured sufficiently to cause an external swelling, bruises or hemorrhage followed by nasal obstruction should be carefully examined, under a general anesthetic if necessary. The septum and

Read before the Sixty-Sixth Annual Meeting of the American Laryngological Association, New York, N. Y., June 7, 1944.

the turbinates should be adequately shrunk with adrenalin and cocaine, and examined for the following: subperichondrial hemorrhage, lacerations of the mucosa, suspicious bends or malposition. I do not find the septal cartilage dislocated from the vomeral groove as frequently as mentioned by Metzenbaum, but when it does occur, if seen early, it can be easily corrected. However, if the condition has existed for more than a week I find it very difficult to set the cartilage back in the groove because of the interposition of newly developed fibrous tissue.

Hematomata are quite common and should be thoroughly evacuated. It is true that in many cases infection will not take place and the blood clot will be absorbed. However, it is not feasible that one run the risk, knowing the destructive effects of a septal abscess, particularly in a child. Besides, there is no question in my mind that the organization of the blood clot leads to fibrosis and interferes with the further growth of the cartilage. Therefore, it is advisable to incise the mucous membrane, evacuate the clot and then pack firmly to prevent further bleeding. The treatment, here, parallels that of hematoma of the auricle.

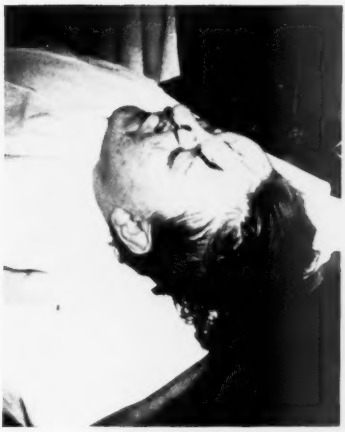
Where the septum is found to be bent or curved, it may or may not be associated with a depression of the nasal bridge, the nasal bones being crowded between the ascending processes of the superior maxilla. X-ray examination may be helpful in determining this question, but external palpation is generally sufficient. In such cases it will not be possible to eliminate the septal curve without at the same time elevating the nasal bridge. This is accomplished by intranasal pressure with a heavy dull elevator or an instrument which I designed for this purpose.[1] The septum can then be pushed back into place and retained by means of packing or a wax plug. In some of these cases it is necessary to apply a copper molded splint over the nasal bridge to overcome the spreading tendency and reshape the immature bones.[2]

Considering now the more extensive injuries to the septum resulting from crushing blows, automobile accidents, a fall from a great height and the impact of missiles, one is confronted with a complex situation which must be analyzed step by step. Having in mind the ultimate restoration of normal breathing channels, therapy is given only at such time and to such an extent as will not interfere with the more urgent indications. Since the complicating factors may far outshadow the septal injury in gravity, these must be given first consideration. Shock and hemorrhage must be con-

trolled, but at the same time the nasal passages may be cleared of blood clots by aspiration and packing inserted to prevent further bleeding, and what is more important, to prevent the aspiration of blood which in a semiconscious or unconscious patient may have serious consequences.

After the patient has been placed in the proper surroundings for more detailed examination and treatment, further therapy will depend on the patient's recovery from shock and the elimination of intracranial injury as a complicating factor. Treatment of a skull fracture, especially when associated with lacerations of the dura, must take precedence over therapy directed toward the correction of the nasal and facial deformities. These may be deferred as long as a week or ten days, if necessary. Nevertheless, the nasal passages can be kept open during this period by means of aspiration and the use of a sterile vaso-constricter solution. The only contraindication to intranasal medication would be a demonstrable cerebrospinal rhinorrhea. The latter, being definite evidence of dural laceration, would mean the necessity for early repair at the hands of the neural surgeon.

The principles involved in the care of recent injuries to the septum are: first, an accurate appraisal of the extent of the trauma; second, the determination of the effect of the trauma on normal nasal function; and third, a plan for the restoration of normal contour together with normal function. The extent of the trauma may vary from a simple fracture or dislocation of the septum to one in which the septum is lacerated, perforated or comminuted with loss of cartilage, mucous membrane and bone fragments. It is extremely important in all cases not only to replace the broken fragments, using sutures in the cartilaginous portion if necessary, but also to see to it that loss of lining mucosa does not lead to crippling contractures which may permanently block the respiratory passages. I show a case illustrating these points (Fig. 1). A young woman was seen within an hour of an automobile accident in which the windshield glass slashed diagonally across the nose cutting through the left ala, the cartilaginous portion of the septum and the columella, into the right nasolabial fold. When I took the first picture before scrubbing, I failed to notice the depth of the laceration, else my photograph would have shown that the lower half of the nose including a portion of the septum was cut completely across, so that the nose could be lifted as on a hinge disclosing the entire interior. It was quite simple in this case to suture the lacerated septum thus preventing overlapping and subsequent depression of the dorsum.



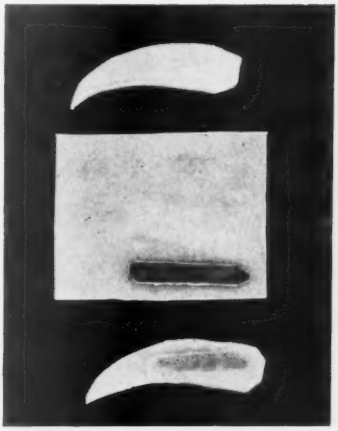


Fig. 1.—Recent laceration cutting across lower half of nose and septum.

Fig. 2.—From above down; wax plug shaped to contour of nasal floor; sheet of dental wax with strip of copper before rolling; wax plug containing copper strip for stiffening.

In another case a man was felled by a heavy box striking him squarely across the nose which was not only crushed, but apparently severed from above downward, remaining attached by the alae. I saw the man for the first time a year or more later, and found that although the nose had been properly sutured in place and had healed (albeit with considerable scarring) he was suffering from an almost complete bilateral atresia due to loss of lining mucosa. After opening up the vestibules (Fig. 2) I found the greater part of his septum absent and the space filled with one of his left turbinates, which had to be removed to clear the airway. It was also necessary to introduce a skin graft to overcome the atresia which was almost complete on the right side.

In simple fractures of the septum it is usually sufficient to replace the broken fragments where they are retained by means of vaseline gauze packs. It is important when introducing these packs to see that they do not displace torn flaps of mucous membrane, that they are evenly inserted so that pressure will be properly distributed, and that they are left in for a long enough period to guard against recurrence of the deformity. I have kept nasal packs in the nose for as long as a week without deleterious effects, except for the

Fig. 3.—Extensive nasal trauma showing partial atresia of vestibule. Enlarged by skin graft.

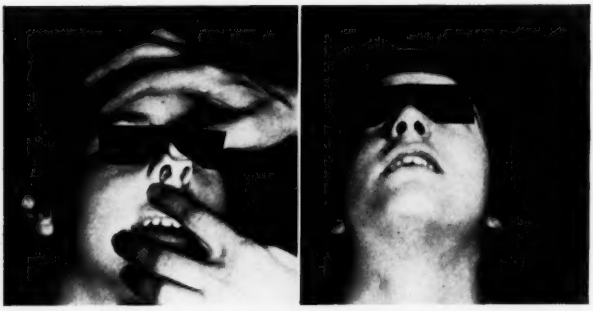


Fig. 4.—Obstruction due to fracture involving the lower end of the septal cartilage. (Patient, aged 13)

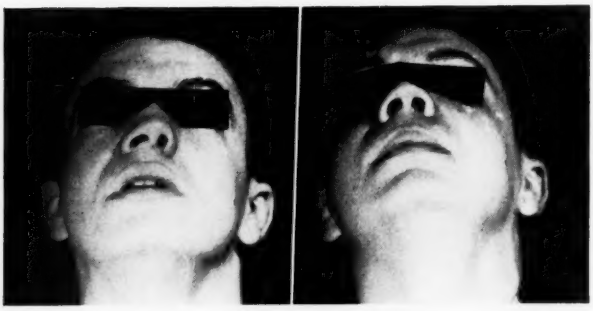


Fig. 5.—Deviation of tip due to septal deformity; dorsum depressed.

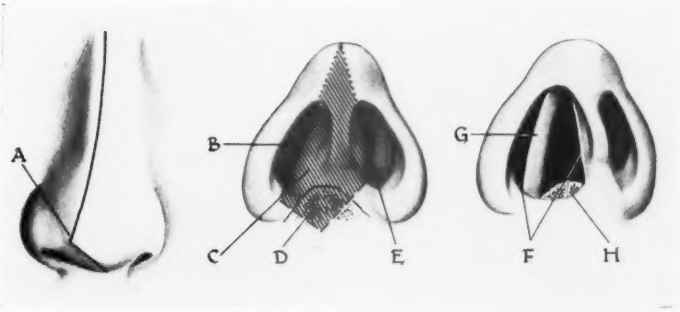


Fig. 6.—Diagram explaining technique in case L.S. (Fig. 4). *A*—angulation lower end of septum. *B*—angle of deviation seen from below; corresponds to *A*. *C*—lower end of septal cartilage blocking right vestibule. *D*—deviated vomer. *E*—septal border projecting into left vestibule. *G*—angle of deviation exposed after removal of deviated cartilage and mucosa. *F*—right and left mucosa as exposed by speculum; only the left mucosa has been freed; the right mucosa is still adherent. *H*—crest of vomer which is removed to permit repositioning of cartilage.

discomfort they caused. Finally, it should be understood that these packs need not necessarily be tight. In most cases the degree of pressure that is required is not as great as is needed, for instance, in the control of a severe nose bleed. Also, it should be borne in mind that severe injuries to the septum are usually associated with fractures of the nasal bridge and other facial bones, and that, therefore, the treatment must be coordinated with all the indications.

Where there has been a loss of septal structure it is extremely important to see that the remaining portions of the septum are kept in the midline. Otherwise, adhesions may so distort the passages as to subsequently interfere with normal nasal respiration. In such cases I have found that paraffin plugs made out of sheet dental wax and properly shaped are well tolerated within the nose for long periods, are easily removed and replaced whenever indicated (Fig. 3.)

Coming now to the septal deformities resulting from inadequate or incomplete primary care, we find that those which give us the most trouble are the ones in which the lower part of the septum has been either dislocated or fractured at an angle causing obstruction on both sides (Fig. 4). Also, there are the cases in which the

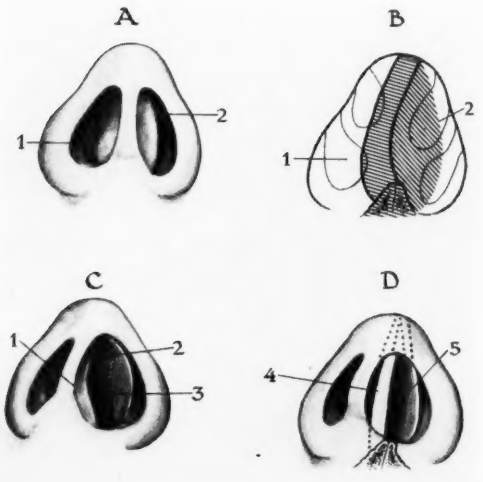


Fig. 7.—Metzenbaum technique. *A1*—angle of deviation; *A2*—exposed free border of septum. *B*—diagram showing relationship of deviated septum and dislocated lower portion of cartilage to the vomer and the turbinates. *C*—lower portion of septal cartilage exposed as far as angle of deviation by dissection of its mucoperichondrium from the right side only; the mucoperichondrium covering the left side of this portion remains attached. 1—Angle of deviation not quite entirely exposed by dissection of mucosa (2). 3—Deviated cartilage to be preserved. *D4*—shows the cartilage severed at the angle of deviation thus liberating the lower deviated portion which can be displaced to the left by the speculum (not shown). This portion is later replaced in the midline after resection of deviation from 4 backwards and upwards.

septum is curved or buckled in the dorsocephalic plane giving rise to marked external deformities (Fig. 4 and 5).

In the first instance if the distance from the free septal border to the angle of deviation is short and the nose itself is sufficiently long, it is much simpler to remove all the cartilage and mucosa below the angle of deviation and by removing corresponding wedges from the alar and triangular cartilages achieve a shortening of the nose. Thus, the angle of deviation becomes the free border of the septum and can be maneuvered back to the midline after removing some of the parallel-lying vomer. This is illustrated in Figs. 4 and 6. In this case the associated kyphosis was reduced at the same time.

Fig. 8.—Scoliosis involving septum and one nasal bone.

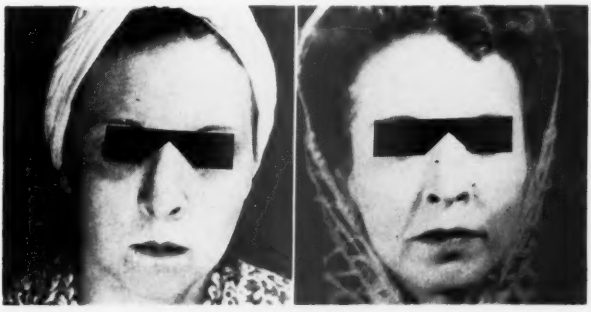


Fig. 9.—Scoliosis involving septum and both nasal bones.

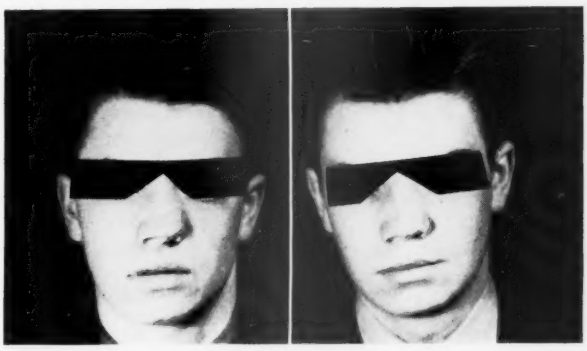


Fig. 10.—Dorsal view of Fig. 5 showing effect of cartilage graft filling dorsal defect.

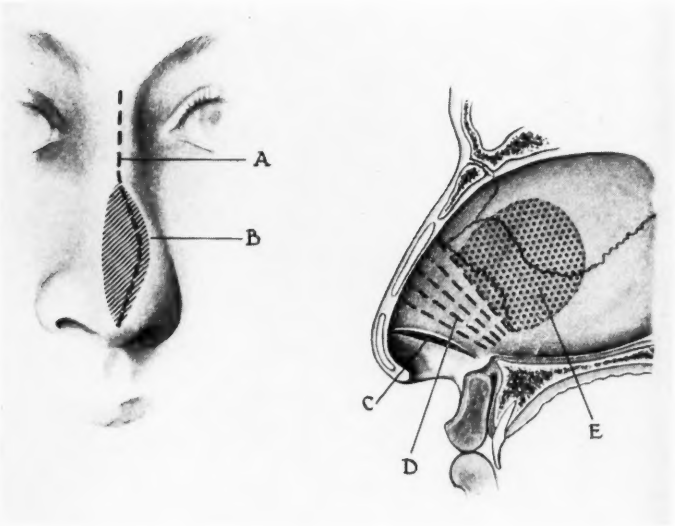


Fig. 11.—Diagram explaining technique in cases shown in Figs. 8 and 9. *A*—dorsal border of septum as visualized and palpated through the skin. *B*—shaded area representing undermining of skin to permit repositioning of cartilage. *C*—incision in septal mucosa. *D*—area of cartilage exposed by dissection of mucoperichondrium showing lines indicating incisions to eliminate the curve. *E*—shaded area representing bone and cartilage removed.

In most cases, however, it is important that the portion of cartilage lying diagonally across both vestibules be preserved, and here the technique devised by Metzenbaum has its place. Difficulty in replacing this portion of the cartilage is encountered at two points, namely, above where the angle of deviation reaches the dorsal skin and below where the cartilage overrides the vomer. Since the objective of the procedure is the preservation of that portion of the cartilage lying below the angle of deviation (Fig. 7), it is important to dissect it free only on one side, leaving the cartilage attached to the opposite mucosa. When the angle of deviation is reached, it must be incised throughout its entire width from dorsal skin down to the nasal floor. This is to facilitate the rotation of the preserved placque back into the midline. In most cases it also is necessary to free the cartilage from its attachment to the vomer. As a rule the cartilage is found overriding the vomer so that replacement is im-

possible unless one of two things is done, namely, the lower or overriding portion of the cartilage may be removed or the vomer taken out. In my experience, it has rarely been possible to lift the cartilage back into position on the crest of the vomer. Finally, having freed the cartilage so that it can readily be swung to the midline without tending to spring back to its former position, it is frequently necessary to separate the columellar cartilages from behind, thus forming a groove into which the free border of the cartilage may be fitted. Naturally, that portion of the deviation behind and above the angle of deviation is resected in the usual manner. The primary incision is closed with one or two silk sutures and the nose packed for 24 to 48 hours.

The second type of deviation which is not amenable to the classic submucous resection is that in which the septal cartilage is buckled or curved away from the midline causing a scoliosis of the nasal bridge. In this type of case we frequently find the entire nasal bridge deviated and it is necessary to refracture the nose bringing the bony arch back to the midline and with it the perpendicular plate of the ethmoid. A submucous resection alone fails to correct either the deformity or the nasal obstruction, because the strip of cartilage which must by this technique be left to support the lower portion of the nose is still curved to one side and holds the mucous membrane like a sheet in its previous position despite removal of considerable underlying cartilage. The procedure which will correct this condition is one in which the cartilage is dissected free on one side only, the dissection being then carried over the dorsal margin directly under the skin which is freed for a distance on both sides forming a pocket into which the cartilage can be pressed after the curve has been eliminated. This is done by making a number of cuts through the width of the cartilage or by removing several narrow strips of cartilage to permit straightening it. Thus, if the nasal bridge is refractured and with it the perpendicular plate brought back to the midline, it will be seen that very little of the septal structure needs to be removed. In most cases the excision takes in the deviated vomer and the sphenoidal process of the quadrangular cartilage which so frequently forms a sharp spur. It is important in these cases to maintain pressure on the previously convex side of the septum for as much as a week or at least until fibrous union has taken place in the hacked cartilage and refractured bones. Figs. 8-11 show the technique described and a typical case.

Occasionally the lower half of the nasal dorsum will be found to be depressed as a result of buckling of the cartilage and no

amount of maneuvering can effectively restore the proper height. In these cases it will be necessary at a later date to insert a cartilage graft to fill the defect. (Fig. 10).

Another type of deformity is one in which the septum has been dislocated from its dorsal attachment and has carried the tip of the nose with it to one side. As a result the nostril on the deviated side is narrowed and if the cartilage were to be removed in its entirety the tip would sag. In these cases it is necessary to dissect the dorsal skin free from the dorsal margin of the cartilage all the way from the free border back to the point where it begins to deviate. If then, at this point, the cartilage is incised throughout its width it will be possible to reposition it. Here too, it will be helpful to groove the columella from behind to permit receiving the free end of the cartilage.

Occasionally one sees a septum which appears to be collapsed and bulging into both vestibules causing obstruction on both sides. This is usually the result of a previous trauma with abscess formation, absorption of cartilage and replacement with dense fibrous tissue. It is necessary in these cases to dissect out all the fibrous tissue and islands of cartilage, indeed a painstaking and tiresome job which can only be safely accomplished with sharp instruments, good hemostasis and illumination. The liberated mucosa of the two sides is then approximated by a through and through mattress suture, reinforced with packing. Subsequently the dorsum is restored and the tip elevated by means of a double cartilage graft.

SUMMARY

1. Trauma to the nose in children should receive careful attention; dislocated septal cartilage should be replaced, hematomata evacuated and spreading of the nasal bones prevented by proper application of an external molded splint.

2. The primary care of injuries to the septum should include hemostasis, replacement of fragments, careful smoothing out of torn mucosa and proper packing. These must be coordinated with the treatment of the other concomitant injuries.

3. Deformities resulting from inadequate primary care must be corrected with due regard for the necessity of preserving such cartilage as is necessary to the support and contour of the nasal bridge and tip.

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XXVII

INTRANASAL VACCINE FOR THE PREVENTION OF COLDS

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AND

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Our previous studies on the use of vaccines for the prevention of colds have yielded disappointing results. The results with two types of oral vaccine were indistinguishable from those obtained with placebos used as controls. Of the cold vaccines injected subcutaneously, one proved entirely without value, while another vaccine caused a reduction in the number of colds, which was of statistical though not of practical significance.[1, 2]

Certain experimental evidence suggests that greater tissue immunity in the nose may be obtained by the local application of vaccine than by the use of the same vaccine administered either subcutaneously or orally.[3-7] In 1935 Walsh[8] reported the results of a preliminary study on the value of intranasal vaccine in the prevention of colds, and in 1940[9] summarized the results of his eight-year study. He concluded that intranasal vaccination against the common cold offers the best means of prophylaxis at our disposal.

During the school year 1941-42 we conducted a controlled experiment to evaluate various vaccines sprayed into the nose as a method of cold prevention. The subjects were university students who were particularly susceptible to colds and who wished to join the "cold prevention group." A total of 479 students were admitted to the study after excluding those whose difficulties seemed to be due primarily to chronic sinusitis or allergic rhinitis, as determined by history and careful examination of the nose and throat. Each student was then assigned without selection to one of four groups. The "vaccine" was dispensed as follows:

From the Students' Health Service, University of Minnesota, Minneapolis, Minnesota.

Group 1. Commercial respiratory vaccine (undenatured bacterial antigen) intended for subcutaneous injection, having the following formula:

Pneumococci	0.0150 mg. N_2 per cc.
Streptococci	0.0150 mg. " " "
H. Influenzae	0.0100 mg. " " "
M. Catarrhalis	0.0025 mg. " " "
Staphylococci	0.0075 mg. " " "

Group 2. Commercial influenza-pneumonia vaccine prepared by the Rosenow method and intended for subcutaneous administration, containing the following:

Pneumococci	1,500 million per cc.
Streptococci (Rosenow)	1,500 " " "
Hemolytic Streptococci	1,000 " " "
Staphylococci (Aureus)	500 " " "
H. Influenzae	500 " " "

Group 3. (Control Group). This was sterile isotonic saline solution containing merthiolate 1:20,000 and just enough fluorescein to render the solution faintly colored and "turbid."

Group 4. Vaccine prepared in the University Hospital bacteriology laboratory by Dr. Milton Levine in accordance with the method utilized by Walsh. The organisms in this vaccine were grown on veal infusion agar slants for 24 hours and were harvested in saline. They were heat-killed at 65° C. for one hour and preserved in merthiolate, 1:20,000. The final product was standardized on the turbidity basis to McFarland tube No. 2. (Approximately 600,000,000 organisms per cc.) Equal parts of the solutions containing the following organisms were used: Pneumococci, encapsulated organisms, types I to XXXII; streptococci, Lancefield groups A, B, C, D, E, F; pathogenic staphylococcus aureus, two strains; neisseria catarrhalis.

The students in all groups were treated in the same manner. Each subject was supplied with vaccine and a DeVilbiss No. 28 nasal atomizer with the following printed instructions:

DIRECTIONS—COLD PREVENTION—1941-42

1. The glass and metal parts of the atomizer are to be sterilized by boiling before the vaccine is put into the atomizer. Fill the atomizer with vaccine, being careful to shake the bottle well. Keep the bottle of vaccine in the refrigerator—the atomizer may be kept in the bathroom closet.

2. Six puffs should be sprayed into each side of the nose each night during the cold season.

3. Do not use the vaccine in the presence of an acute cold.

4. Be sure to shake the atomizer before using.

5. If you develop a cold in spite of the preventive treatment, report immediately to the Health Service Dispensary for treatment.

6. Each week a card will be put in your post office box. Please fill this card out promptly and accurately and drop it in the "campus mail." This is our method of keeping accurate records of your "cold experience" during the time you are taking the vaccine—roughly, from October 15 to May 1.

7. If you lose these directions, please ask for another set.

8. Be sure to apply at the Health Service desk for a "refill" of your vaccine when your supply gets low, so that you will not miss any treatments.

Once a week each student found in his post office box a report card which he was required to fill out and return to the Health Service. In addition, each member of the group was seen at the end of the study for a check on the accuracy of reporting. The content of the report cards and the method of assembling the data have been reported elsewhere[10] and will not be repeated here.

RESULTS

Of the 479 students who enrolled in the cold prevention group in the fall of 1941, only 321 satisfactorily completed the study. This was due largely to the fact that the plans of many students were changed because of the war, although a few found the requirements of a weekly report too trying on their spirit of cooperation. Some students were dropped when it became apparent from their report cards that they had perennial rhinitis which varied very little from month to month. The results in the table are taken only from the records of those who completed the study.

It is obvious from the data that although the vaccinated groups showed a reduction in the number of colds during the study as compared to the usual number of colds they reportedly had had previous-

RESULTS WITH INTRANASAL COLD VACCINES

1941-1942

	1	2	3	4
Subjects who began study	123	124	109	123
Subjects who completed study	86	78	72	85
Percentage who completed study	70	63	66	69
*No. colds per person during previous year—average	5.3	5.3	4.8	5.0
No. colds per person during year of study—average	2.3	2.4	2.5	2.4
Percentage of group with no colds during year of study	4.7	6.4	8.3	8.2
No. days per person lost from school—average	1.2	1.2	1.0	1.3
% of students with colds in which complications developed	23.2	16.4	13.6	17.9
Percentage of students hospitalized with complications	1.2	1.4	3.0	3.8
Classifications of colds experienced (% of total colds in group)				
1. Student's opinion as to severity:				
Mild	44.6	44.4	41.0	48.5
Moderately severe	32.6	33.9	36.5	28.2
Severe	15.0	15.3	16.9	18.8
Very severe	7.8	6.3	5.7	4.5
2. Based on symptomatology:				
Mild	74.7	73.7	76.8	80.2
Moderately severe	15.7	18.4	14.1	12.4
Severe	5.1	4.7	6.8	3.5
Very severe	4.5	3.2	2.3	4.0
3. Based on student's opinion as to comparison with previous colds:				
Very much milder than average previous cold	23.0	27.4	24.0	24.7
Somewhat milder than average previous cold	37.0	35.4	32.0	34.5
About same as average previous cold	27.3	31.1	35.3	29.9
More severe than average previous cold	11.5	4.3	7.3	9.8
Much more severe than average previous cold	1.2	1.8	1.3	1.1
4. Average Duration of colds, days	7.7	7.8	7.8	7.3

Group 1. Commercial Respiratory Vaccine
Group 2. Commercial Influenza-Pneumonia Vaccine
Group 3. Control (Saline Solution)
Group 4. Fresh Vaccine

*Reported from memory.

ly, the same was true of those in the control group and no significant difference exists between the experimental and the control groups. The percentage of students who had no colds during the year and the number of days lost from school were essentially the same for all groups. This was also true in regard to complications following colds (such as sinusitis and otitis). The colds that did occur were apparently as severe and as prolonged among the vaccinated groups as among the controls.

CONCLUSIONS

This controlled study furnishes no evidence that intranasal vaccine is effective in reducing the number or the severity of colds.

UNIVERSITY OF MINNESOTA.

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XXVIII

AEROSINUSITIS—ITS CAUSE, COURSE, AND TREATMENT

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The great increase in aerial transport and the widespread use of low pressure chambers for the indoctrination and classification of flying personnel which has occurred during the present world conflict has focused attention upon certain conditions produced by the more or less rapid changes in barometric pressure, consequent to ascent and descent. From the viewpoint of the otorhinolaryngologist, the most important of these conditions is aero-otitis media. However, another condition, aerosinusitis, is a very definite entity and may at times be quite spectacular. In the experience of this observer, aerosinusitis presents an incidence of about one-twentieth that of aero-otitis media and occurs under similar conditions.

A description of this entity which only recently came to be known as aerosinusitis[5] was first offered by Marchoux and Nepper in 1919,[8] who after numerous experiments in the Paul Bert Bell wrote the following:

> "Our observations lead us to believe that great importance is to be attached to the integrity of the rhinopharyngeal mucosa. Any obstruction which prevents the rapid establishment of pressure equilibrium between the cranial air spaces and the outside atmosphere involves the possibility of a series of misfortunes in which pain is one of the least and syncope one of the most serious. All become more marked as the pilot approaches land and at a time when he needs his faculties for a safe landing. While the atmospheric pressure decreases by 10 cm. of mercury between sea level and an altitude of 1,000 meters, it does not vary more than 4 cm. between 6,000 and 7,000 meters. It is therefore in the lower layers of the atmosphere that the defect is most felt."

Since that description, a number of investigators, including Behnke and Willmon,[2] Brouwer,[4] Pastore,[9] Von Diringshofen,[12] Hermann,[6, 7] Salem,[11] this investigator,[5] and many others have concerned themselves with certain aspects of the condition. However, very little has been written relative to diagnosis, clinical course, or treatment.

Presented as a Candidate's Thesis to the American Laryngological, Rhinological and Otological Society.

Aerosinusitis is an acute or chronic inflammation of one or more of the nasal accessory sinuses produced by a barometric pressure difference between the air or gas inside the sinus and that of the surrounding atmosphere. It is commonly characterized by congestion and inflammation of the lining structures. Pain over the area of the sinuses is usually present. Mucosal or submucosal hemorrhage may occur. The condition may at times result in temporary or permanent change in the mucous membranes lining the sinuses depending upon the amount of barotrauma involved.

The dynamics of the production of aerosinusitis have been discussed in a previous work.[5] It may be summarized as follows: The density of the atmosphere decreases with altitude in accordance with the following table:[5]

ALTITUDE, IN FEET	PRESSURE, IN MILLIMETERS OF MERCURY
Sea Level	760.0
5,000	632.4
10,000	522.6
20,000	349.2
30,000	225.6
40,000	140.7
50,000	87.3
60,000	54.1
70,000	33.6
80,000	20.8

A cavity (such as a sinus) will, when moved from an environment of relatively high barometric pressure to one of relatively low barometric pressure, as in ascent, undergo alteration of itself or its contents in such a manner that equilibrium between the gas or air inside the cavity and that outside will be attained. During descent from an environment of relatively low barometric pressure to one of relatively high barometric pressure, alteration also takes place, but the response is to forces acting in an opposite direction.

In the instance of a normal sinus with an unobstructed ostium, free flow of gas or air between the cavity of the sinus and the outside environment brings about equilibrium during ascent and descent without change in structure and consequently without sensation.

Two conditions, however, may alter or prevent this free exchange of the gaseous matter. They are, first, the presence of fluid, mucus, pus, or similar substances covering the ostium, and second, obstruction of the ostium by redundant tissue or anatomical deformity. The first instance represents the basic pathology for the develop-

ment of *non-obstructive aerosinusitis* and the second the basic pathology for the development of *obstructive aerosinusitis.*

Non-Obstructive Aerosinusitis. When there is fluid, pus, or mucus covering the ostium in such a manner that it may be pushed away by relatively small pressure changes, little happens on ascent as the flow is outward.* On descent, however, the direction of flow is reversed and fluid, mucus, or pus may be pressed into the sinus. Usually this phenomenon takes place without pain or other sensation but it represents a mechanism by which an uninfected sinus under certain circumstances may become infected.

If purulent sinusitis occurs under these circumstances, it does not differ from any other sinus infection, and except in those instances presenting a history of lengthy descent during the existence of a productive cold, cannot be differentiated from the usual type of sinusitis.†

Obstructive Aerosinusitis. The circumstances are quite different when the ostium is blocked by swollen, or redundant, tissues or anatomical deformity. In this instance the air or gaseous contents of the cavity are trapped and during altitudinal change produce a pressure, positive in ascent or negative in descent, relative to the environmental pressure.

The trapping effect is believed to be due to a flutter or ball valve action. It is usually produced only during descent, when inflamed tissue, a swollen turbinate, or a polyp is pressed or sucked (depending upon your point of view) against or into an ostium in such a manner as to produce an airtight seal. Viscous secretions probably augment the seal. Rarely, the trapping effect occurs on ascent. Although the pathology has never been actually observed, it would seem reasonable to assume that in these rare instances a ball or flutter valve was being formed by a structure inside of the sinus. A polyp or a bit of redundant tissue could be the causative factor.

Equalization of the pressure of the gas inside the sinus and that outside, when there is obstruction of the orifice, necessitates certain

*In fact, it is not at all a rare occurrence for a sinus to empty itself of old fluid or pus during ascent. In this connection, Andrews, Roth, and Ivy[1] have reported temporary relief from acute symptoms in some of their patients who were treated for sinusitis by means of pressure chamber excursions.

†It is interesting to speculate why purulent sinusitis produced in this manner is not a much more common occurrence than it is in fact. Certainly many people are flying today and in spite of admonitions many fly with productive colds. It is the belief of this investigator that two factors are involved. First of all, the amount of material which is present to be pressed into a sinus is relatively small. Second, healthy, untraumatized epithelium is not easily infected.

alterations of structure. The bony or cartilaginous walls of the sinus are elastic only to a very minor degree. Consequently, compensation cannot take place by expansion or contraction of the external walls.

The only elastic material which can alter the size of the cavity is the lining membrane. The only other space-filling mechanism by which equalization can be fostered is fluid production, either mucus, blood, or serum.

As has been stated, blockage on ascent is the exception. The occasional patient reports pain over a sinus after a certain degree of ascent. Return to earth or to lower levels affords relief. Even remaining at altitude is usually not accompanied by much difficulty, as the gases under pressure are rapidly absorbed to the point of pressure equalization.

On the other hand blockage during descent is the more common occurrence. Under such circumstances, the relative negative pressure (vacuum) must be compensated for and alteration in structure and contents follows. The degree of alteration depends upon the degree of pressure differential. If the degree is minor, adequate compensation will be afforded by increased secretion of fluid to fill sufficient space to equalize the pressure and release the flutter valve if such a mechanism was the causative agent. This condition may be labeled *first degree obstructive aerosinusitis*, and is characterized by only slight, if any, pain, and usually insufficient symptoms to come to the attention of the physician.

Second degree obstructive aerosinusitis could readily be the designation of the condition when characterized by localized or generalized swelling of the mucosa with exudation of tissue fluid. This degree of involvement produces definite symptoms and findings. Pain and hyperesthesia over the sinus are present, and remain for some time (one to seven days) after descent. There is usually only slight, if any, fever or leukocytosis. Roentgenography demonstrates thickening of the lining membrane and possibly some clouding produced by fluid.

The most severe degree of obstructive aerosinusitis may for clarification be categorized as that of *third degree aerosinusitis*. The differential pressure necessary to produce such a condition must be relatively great—18,000 feet or more. The resultant picture is that of extensive swelling of the lining membranes, often accompanied by extravasation of blood into the cavity of the sinus or submucosal hematoma. Stripping of the membranes from the bony wall has been reported.[6,7] In this instance, trauma is great, and in at least one case seen

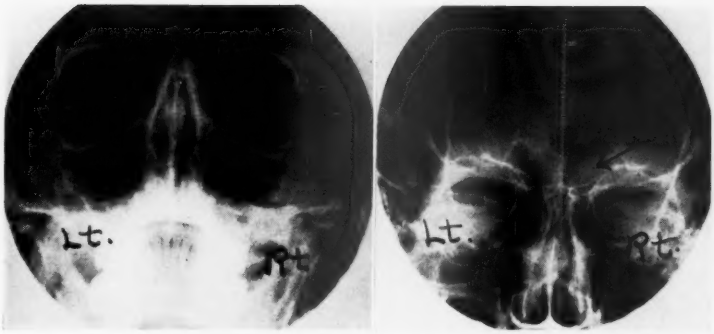

Fig. 1.—X-ray pictures demonstrating submucosal hematoma of floor of right frontal sinus (Case 1).

by this investigator a fulminating infection followed, which caused no little concern.

Excruciating pain over and about the affected sinus is present. The sensation has been described by airmen to resemble a bee sting. Fever and leukocytosis are present. Roentgenography demonstrates thickened lining, clouding by fluid or blood, and at times, submucosal hematoma, or stripping of the lining membrane. Resolution, if complete, takes from 7 to 21 days. It is probable that in at least some of these instances the insulted tissues never return to normal.

It is interesting to divert at this juncture to point out that epistaxis occurring during altitude excursions may be due to this mechanism. In such a case the flow of blood into the sinus will fill sufficient space to bring about an equalization of pressure, releasing the block. After release of the block the blood will flow out into the nasal cavity.

Sinuses Involved. The frontal sinus seems to be involved much more often than any of the other sinuses. Incidence of involvement of the maxillary sinuses offers a poor second. Aerosinusitis of the other sinuses is very rare, or at least the complaints are so slight or so vague that they do not come to the attention of the otorhinolaryngologist. Four probable reasons may be given. First of all, the length and the structure of the nasal frontal duct offer much more opportunity for obstruction. Second, large sinuses are more often affected than the smaller sinuses. In view of the dynamics of production, this would seem reasonable to expect. Third, the position of the ostium of the

frontal sinus would lead one to expect occasional flutter valve action of the turbinates, and redundant or swollen tissue of the turbinates. Fourth, the frontal sinuses have no accessory openings.

REPORT OF CASES

CASE 1: On February 25, 1943, Lt. A had a slight cold, with a moderate amount of discharge in his nose. He ascended to more than 10,000 feet in an airplane and engaged in violent acrobatics during which his altitude varied from 10,000 to 7,000 feet. There were no subjective symptoms during this flight although he nearly "blacked out" once or twice, due to the violence of the maneuvers. After returning to the ground, he decided to go up with another pilot. They ascended to 5,500 feet and then began to descend in a power-on let-down at a rather rapid rate. During this let-down Lt. A suddenly developed a sensation as if a bee had stung him just above his right eye. The impression was so distinct that he was surprised when his hand did not encounter an insect in the area, and to make certain he checked the lining of his helmet for any rough surface which might have produced the sensation. The aircraft was leveled off, and then descent was continued more slowly. The stinging sensation then became a localized pain, extending laterally along the region of the eyebrow and then to the lower rim of the orbit. By the time ground level was reached the pain had become very acute.

In order to relieve the discomfort, Lt. A then re-ascended, and when he became more comfortable started to descend slowly. The pain re-appeared, and becoming disgusted Lt. A dived the aircraft and was surprised to find that the pain did not increase further. The use of the Valsalva maneuver gave relief from the acute symptoms and when he landed only a dull ache was present.

Examination of the nasal cavity by a competent otolaryngologist revealed no gross findings, with the exception of a slight mucoid discharge.

Roentgenologic examination demonstrated a localized dome-shaped swelling of the mucosa of the floor of the right frontal sinus (Fig. 1).

The flier was grounded for one day only and subsequently flew at low levels. The aching sensation along the left eye continued for about three days, but there was no occurrence of acute symptoms. No serosanguineous discharge was present at any time, nor was there any increase or change in the character of the existing nasal discharge.

Unfortunately, this officer was transferred while radiologic evidence of this pathology still existed.

Diagnosis. Third degree obstructive aerosinusitis of the right frontal sinus with submucosal hematoma.

CASE 2. On August 11, 1943, Corp. J. ascended to 38,000 feet in a low pressure chamber where he remained for a short period of time. Descent was made at a rate of 6,000 feet per minute. When the level of 26,000 feet had been reached, he suddenly noticed a sharp pain in his left supra-orbital region. The sensation was described as feeling as though something had burst over and to the nasal side of his eye. The pain was relieved slowly by leveling off and then re-ascending about 1,000 feet. Descent was then begun. During the descent it was necessary, because of pain, to level off five or six times before reaching ground level. There were no painful aftereffects. However, an x-ray picture of his sinuses was taken (Fig. 2), which on comparison with a routine x-ray picture taken July 6, a little over a month before, demonstrated a large dome-shaped swelling of mucous membrane in the left frontal sinus, which had not been present on July 6. At no time was there any bleeding from the nose. There were no complaints of nasal obstruction.

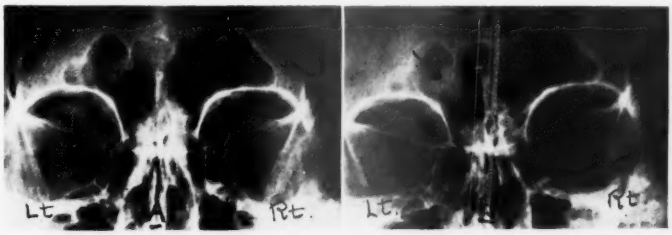

Fig. 2.—X-ray pictures demonstrating (a) normal sinuses taken July 6, 1943; (b) x-ray findings of submucosal hematoma following barotrauma received August 11, 1943 (Case 2).

Past history disclosed periodic attacks of sinusitis in the spring of the year since the age of 15. He had been under treatment for sinusitis once or twice a year since the age of 15.

Diagnosis. Third degree aerosinusitis with submucosal hematoma.

CASE 3. On May 26, 1943, Corp. E ascended in a low pressure chamber to a level of 30,000 feet. On descent to 24,000 feet he experienced pain over his left eye, which was partially relieved by stopping descent and re-ascending 2,000 feet. Descent was then resumed but had to be interrupted several times to relieve the increased sinus pain. Slight pain and sensitivity of the left upper teeth developed during this procedure. On reaching ground level, the entire left side of the subject's face was painful. This was relieved after a short time at ground level, and a serosanguineous discharge appeared in the nasal cavity. The pain did not recur, but on arising the following two mornings a large amount of serosanguineous matter was blown from the nose.

Corp. E had had no recent cold before ascending in the low pressure chamber. He had been sneezing a few times each morning and had had symptoms of hay fever several years ago. Examination of the nasal mucous membrane demonstrated slight inflammation on the left side. No discharge was present. The airways were adequate. Transillumination revealed extreme opacity of the left frontal sinus and a slight decrease in light transmitted through the left maxillary sinus. Roentgenography findings (Fig. 3) demonstrated haziness of the left antrum and the left frontal sinus, which was attributed to mucosal thickening. The remaining sinuses were normal.

On June 4, 1943, Corp. E reported that he had had a serosanguineous discharge from his nose for the past three days. He was free from pain or any sinus complaints. The left antrum was clear upon transillumination. The left frontal sinus transmitted more light than it had on the previous examination. Recheck of roentgenographic findings (Fig. 3) showed improved radiance in the left frontal and maxillary sinuses, as compared to the examination which had been previously made.

Diagnosis. Third degree obstructive aerosinusitis of the left frontal sinus, second degree obstructive aerosinusitis of the left antrum.

CASE 4. Pvt. R ascended in a low pressure chamber at the rate of 7,000 to 8,000 feet per minute to 38,000 feet. He remained at that altitude for 22 minutes, and descended at a rate of 5,000 feet per minute. During descent he had no pain in either ears or sinuses. After being at ground level for about five minutes he suddenly developed a throbbing pain in the right supra-orbital region associated with blurring of vision. The pain became more intense over a period

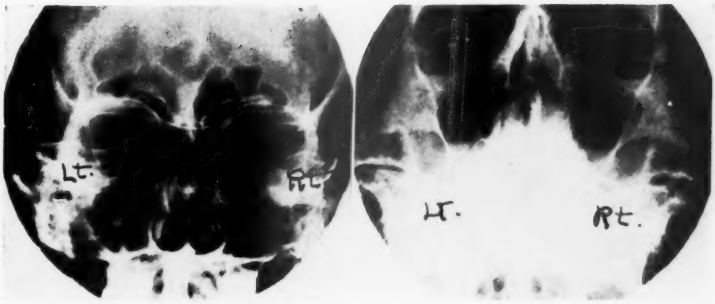

Fig. 3.—X-ray pictures demonstrating obstructive aerosinusitis of the left frontal and maxillary sinuses (Case 3).

of about one-half hour, then remained at about the same intensity. Two hours and twenty-five minutes after reaching ground level the patient reported to the Ear, Nose and Throat Clinic, suffering from a throbbing pain in the supra-orbital region, which was unrelieved by 20 grains of aspirin. Transillumination of the right frontal sinus demonstrated practically no transmission of light. Roentgenographic study disclosed increase in the thickness of the membranes and a haziness over all. One per cent ephedrine solution was then sprayed into and over the region of the right semilunar hiatus, which brought about marked relief. On gently blowing his nose, a quantity of serosanguineous discharge appeared. Subsequent transillumination of the right frontal sinus demonstrated that it was practically clear. Complete resolution was present at the time of an examination seven days later.

Diagnosis. Third degree obstructive aerosinusitis of the right frontal sinus.

CASE 5. Second Lt. M stated that on the afternoon of May 13, 1943, he had been flying at an altitude of about 5,500 feet. Descent was made at the rate of about 1,000 feet per minute, and during descent he began to have severe pain over both frontal sinuses. At ground level the pain was markedly severe; consequently he reported to his surgeon, who made a diagnosis of aerosinusitis. Three per cent ephedrine nose drops were introduced into the nose at once, which produced temporary relief. During the night, however, the frontal sinus pain became more severe, and on May 14 he again reported to his surgeon. His surgeon recommended that he be placed

in a low pressure chamber and ascended. At 6,000 feet the pain had entirely disappeared. The chamber ascent was then continued for an additional 3,000 feet, which was followed by a slow descent of 5,000 feet where the pain recurred. Re-ascent of 3,000 feet was then made, and descent started at a rate of 3,000 feet per minute. After a series of these step-like descents, the patient arrived at ground level with his pain entirely relieved. He was examined three days later. There had been no recurrence or discomfort of any sort.

Past history. The patient admitted having had an acute cold about two weeks previous to this experience.

Diagnosis. Obstructive aerosinusitis of both frontal sinuses, degree undetermined.

TREATMENT

The treatment of aerosinusitis, as is the case in any other type of sinusitis, is primarily dependent upon the good judgment of the otorhinolaryngologist, who must weigh and consider the various aspects of the situation. He must study the mode of origin, the mechanical forces of production, and then attempt to reverse the existing process with a minimum of trauma to the already barotraumatized tissues.

Non-obstructive aerosinusitis in most instances responds to the simplest forms of treatment, if any treatment is necessary. Shrinkage of the structures which might obstruct normal drainage will usually allow normal ciliary activity to evacuate the materials which have been pressed into the sinus. The use of external heat seems reasonable.

The obstructive types present a somewhat different problem. In this instance, the primary attack must be directed toward equalizing the pressures inside and outside the cavities. The ideal treatment would seem to be a return to the altitude at which the block was formed, correcting the obstruction, and then a slow return to ground level. This can be accomplished either in a low pressure chamber or by actual flight, and has given good results in some cases. However, one should know the exact pathology before such a procedure is instituted, as submucosal hematomas, stripping of the lining membranes and frank hemorrhage into the cavity of the sinuses will not be benefited by the changes in barometric pressure taking place during such an excursion, and they may be exaggerated.

Carefully calculated and calibrated Proetz[10] treatment has been used in two instances by this observer with seemingly good results.

With such a procedure, however, one must attempt to use only sufficient negative pressure to overcome the block. One must be fearful of introducing infected materials into the traumatized tissues which are present. More cases must be studied before this procedure can be advocated.

Strictly conservative methods to aid space-filling fluid production by the lining membranes have in most cases been sufficient, although the time of recovery may be slightly lengthened. Heat and shrinkage or mechanical movement of the tissue forming the block seem to bring about this result. As soon as sufficient fluid has formed in the cavity of the sinus, pressure is equalized, the valve is released, and drainage and ventilation may take place, followed by the normal healing processes. Critical judgment must be used in selecting the time of return to flying duties of personnel who have developed aerosinusitis.

CONCLUSIONS

1. Aerosinusitis is a definite entity.
2. Aerosinusitis has been defined and categorized.
3. The dynamics of production have been reviewed.
4. Five case résumés have been presented.
5. Treatment has been discussed.

Acknowledgment is made to Lt. Fred W. Ogden, M.C., Capt. Ben H. Senturia, M.C., and Capt. Richard M. Leick, M.C., for aid in the gathering of material for this study.

THE SCHOOL OF AVIATION MEDICINE.

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XXIX

EXTRALARYNGEAL SURGICAL APPROACH FOR ARYTENOIDECTOMY. BILATERAL ABDUCTOR PARALYSIS OF THE LARYNX

HENRY BOYLAN ORTON, M.D.

NEWARK, N. J.

Laryngologists have long recognized that surgery of the neck, especially thyroidectomies, is one of the most frequent causes of bilateral abductor paralysis of the larynx. Other conditions may also cause this condition.

Surgeons and laryngologists for years have been devising methods whereby these patients could have relief from their condition. Today, some form of arytenoidectomy, with fixation of the cord laterally, seems to be the operative procedure. I wish to describe an extralaryngeal approach to the arytenoid.

Knowledge of the part in question is necessary to allow the surgeon access with the least disturbance, and to enable him to carry out his task with as little effect on function as possible.

Trotter[1, 2] after more than 20 years of experience, established the operation of lateral pharyngotomy by the transthyroid route, as a method of general usefulness in surgery of the pharynx and laryngopharynx. Unfortunately, this method or slight modifications thereof are not used as frequently as they should be; for instance, removing the lateral half of the hyoid bone enables one to have perfect access in removing a neurofibroma of the pharynx without contamination of the mucous membrane of the pharynx. Then by removal of the cornua of the hyoid bone and the lateral thyroid cartilage one has access to the laryngopharynx. And now removal of the posterior half of the thyroid ala gives adequate access to the arytenoid without entering the pharynx or the larynx. The first step in the actual attack on the arytenoid itself must be proper exposure. Application of this procedure is now described.

Read before the Sixty-Sixth Annual Meeting of the American Laryngological Association, New York, N. Y., June 8, 1944.

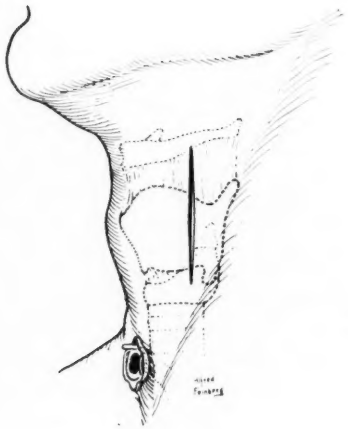

Fig. 1.—Shows the longitudinal incision over the lateral half of the thyroid ala.

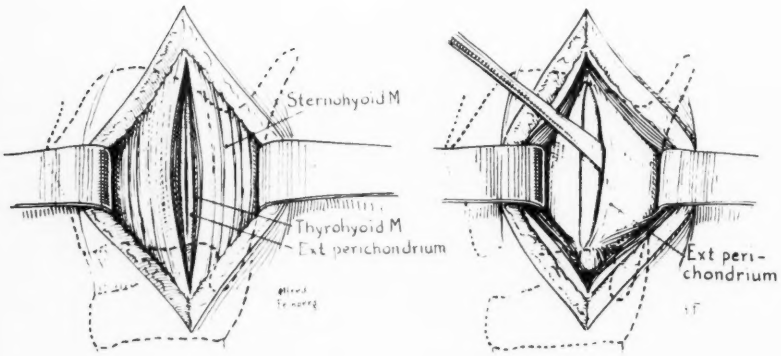

Fig. 2.—Splitting of the sternohyoid and thyrohyoid muscles and incision of the external perichondrium in line of incision.

Fig. 3.—Elevation of the external perichondrium from the posterior half of the thyroid ala. This separation is carried around the under surface of same.

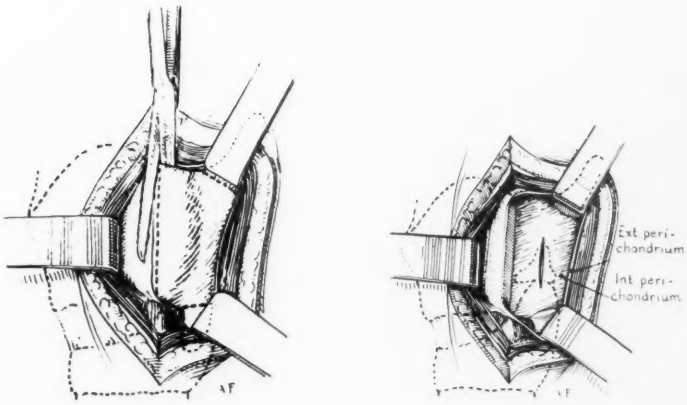

Fig. 4.—Shows the perichondrium entirely separated and the posterior portion of the thyroid ala being removed by heavy shears.

Fig. 5.—Shows the internal perichondrium and the pharyngeal aponeurosis, and by palpation the arytenoid can be definitely identified. It also shows the incision over the arytenoid through its muscular attachments.

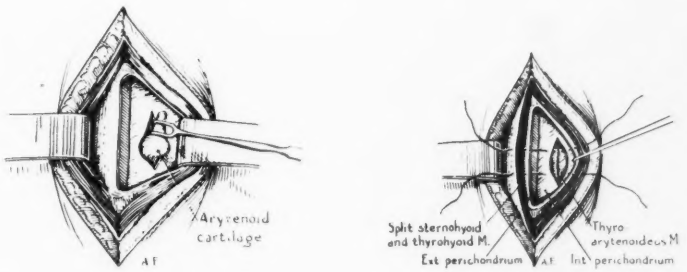

Fig. 6.—Shows the arytenoid cartilage being removed from its muscular attachments.

Fig. 7.—Shows the insertion of sutures, which has the tendency of pulling outward that side of the larynx.

LATERAL TRANSTHYROID APPROACH

The position of the patient is the same as for a complete block dissection of the neck. The chin is rotated laterally and a small sand bag or pillow is placed under the shoulder of the side to be operated on; this position gives a flat surface to that side of the neck.

The anesthesia may be either avertin, ether, or local, depending on the operator.

After proper preparation of the surgical field, a longitudinal incision is made over the lateral half of the thyroid ala on the side to be operated on, extending downward from a point midway between the hyoid bone and the thyroid cartilage to the level of the cricoid cartilage. This incision is through skin and platysma. The sternohyoid, thyrohyoid and sternothyroid muscles are split in the line of incision. The omohyoid muscle is lateral to the incision. With retraction laterally and mesially of the muscles, the perichondrium is then incised in the line of incision; the perichondrium with attachments of the inferior constrictor muscle is elevated from the lateral half of the thyroid cartilage. This separation is carried around the under surface of the thyroid cartilage. With complete separation of the thyroid cartilage from the external and internal perichondrium, the posterior lateral half of the thyroid ala is then removed by means of heavy scissors.

The removal of this portion of thyroid cartilage exposes the laryngopharyngeal aponeurosis and then by palpation at the lower angle of the wound the arytenoid is located. Incision is then made around the internal perichondrium on to the arytenoid cartilage, then by sharp dissection the entire arytenoid cartilage is freed from its muscular attachments and removed.

Two or three fine chromic catgut sutures are placed through the thyro-arytenoideus muscle to the external perichondrium and the anterior split sternothyroid and thyrohyoid muscles. A small cigarette drain is inserted, the fascia closed by two or three interrupted sutures and the skin closed.

This cigarette drain is removed in three or four days. No feeding tube is used.

SUMMARY

The advantages of the procedure herein described are:

1. Access to a wider field of operation, because of the lateral transthyroid approach.

2. The longitudinal incision and splitting the sternohyoid and thyrohyoid instead of the transverse incision.

3. Direct identification of the arytenoid cartilage by palpation beneath the laryngopharyngeal aponeurosis.

4. Complete visibility of the arytenoid during the entire arytenoidectomy.

5. Direct extralaryngeal surgical attack to the side of the larynx.

The procedure has proven very satisfactory and from letters I have received from other men who have followed this procedure their results have been very gratifying; they comment upon the ease with which the operation can be accomplished.

I feel quite certain the approach is sound and time alone will tell if arytenoidectomy is the solution to the problem of bilateral abductor paralysis.

224 DELAVAN AVENUE.

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XXX

TEMPORAL ARTERITIS

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My paper is based upon a clinical observation of two cases, plus a careful scrutiny of all the case reports which have appeared in the literature. I wish, therefore, to present a composite picture which evolved from this type of research work. In this analysis there are 13 papers pertaining to this subject, in which 19 case reports are presented. I must state at this time that Horton and his associates at the Mayo Clinic, who introduced this term, "Temporal Arteritis," have six case reports in this presentation. However, in a personal communication from Horton (December 23, 1943) he states that they have observed 14 additional cases which have not been reported in detail but "that will be done in the near future." We have, hence, 19 case reports to work with, all of which appear as a digest in the appendix to my paper. The composite picture I refer to is as follows:

The patient, most likely a white woman around sixty years of age or over, complains of severe pain in the temporal region. Her internist will tell you that the patient has a fever, leucocytosis, general malaise, and sweats. Every known test has been made and found negative. The internist hopes you will locate a sinusitis which is causing the severe pain and general symptoms. You make your examination, find no sinusitis, and are unable to relieve the pain by cocainization of the ethmoid and sphenoid areas. The days drag along and then in from two to six weeks a bright red, tortuous swelling of the temporal artery appears. The patient is then informed that she has a "temporal arteritis" which will cause symptoms for many weeks or months and then gradually subside. Thus far, no fatal case has been reported. You must bear in mind, however, that in the reports we find that eight patients had an associated retinal arteritis with permanent diminution of vision or complete blindness. That is a high incidence.

I do not wish to infer from the above that the rhinologist will treat the patient daily until the swelling of the temporal artery ap-

From the Research Department, Santa Barbara Clinic, Santa Barbara, Cal.
Read before the Western Section Meeting, American Laryngological, Rhinological and Otological Society, Los Angeles, Cal., Jan. 22, 1944.

pears about a month later; but I do believe that we should include this syndrome in our differential diagnosis. Furthermore, I believe that we should learn all that is known about a subject which we must incorporate in our differential diagnosis. That is the object in presenting this paper.

THE LITERATURE

In 1934, Horton, Magath, and Brown[1] of the Mayo Clinic published an article in which they introduced the term, "temporal arteritis," and reported two cases. In February, 1937, MacDonald and Moser[2] of Indianapolis reported a case and used this new term. In September of 1937, Horton and Magath[3] refer to these first three case reports, present another case report, state that they had seen two other similar cases, and had personal communications from three other doctors, including one from Uruguay. As a result of these nine cases they concluded, "These reports confirm our original impression that we are dealing with a new clinical syndrome."

In 1938, Jennings and Camb[4] of London had an article in *Lancet* entitled "Arteritis of the Temporal Arteries" and reported two cases. However, the apparent object of this exhaustive paper is to question the statement that temporal arteritis is a distinct clinical entity. They believe that this is the same form of arteritis which may affect other arteries. They, therefore, concluded their paper with the statement, "The temporal arteries are, after all, the only really prominent arteries, and if they are spared during arterial inflammation, the nature of the illness may be entirely misunderstood. A subacute or chronic arteritis in other vessels may have been overlooked."

In the next issue of *Lancet*, 1938, Bain[5] gives a brief report of one case. In February 1940, Dick and Freeman[6] of Chicago reported two cases. They disagree with Jennings and Camb and are convinced that temporal arteritis is an individual entity, but fail to explain the loss of vision in each case. In August 1940, Bowers[7] of Seattle reported a case. He states that there are many points in favor of designating this condition as a separate entity; however, he does illustrate that in his case the patient first had an occipital arteritis, five weeks later a temporal arteritis, and a year later some form of a cerebral arteritis, and recovered from each attack.

In December 1940 Sprague and MacKenzie[8] presented a case in which the patient had a bilateral temporal arteritis, marked systemic reaction, and a cerebral arteritis which simulated an encephalitis. In fact, the temporal arteritis had subsided at their last

observation and they stated that "the possibility of permanent cerebral involvement is suggested in this case."

The next case report to appear in the literature entitled, "Temporal Arteritis," was presented by Scott and Maxwell[9] of Lexington, Kentucky. According to this report, the onset was with an arteritis in both arms, followed by an occipital arteritis, and then a bilateral temporal arteritis. Relief of symptoms followed resection of segments of each temporal artery. However, two months later the patient had loss of vision in the right eye and some diminution in the left.

In September 1942 Schaefer and Sanders[10] of Kansas City reported a case in which the pains and headaches were so severe that no sedation would help. In fact, the patient became desperately ill, speech became blurred, and a question of a brain tumor arose. Finally the patient was put on the Sanders oscillating bed, and enjoyed her first symptomless and natural sleep without medication. This oscillating bed was used for seven weeks and the patient was cured.

The eleventh article to appear in the literature on this subject is by Murphy[11] of New York City in December 1942. He states, "Excision of a section of the superficial temporal artery is the only therapeutic procedure generally advocated."

The last paper appeared in February 1943, by Johnson, Harley, and Horton[12] of the Mayo Clinic and is entitled, "Arteritis of the Temporal Vessels with Loss of Vision." They refer to the fact that diminution of vision was noted in four previous case reports and they present three additional ones observed by them and a communication from Reuben Johnson. Their explanation of the involvement of the retinal artery is based upon a paper of Kershner which appeared as a thesis at the University of Minnesota in 1940 entitled, "Blood Supply of the Visual Pathway." According to this, "the anterior deep temporal arteries have an anastomosis with the lacrimal branch of the ophthalmic artery. The ophthalmic artery gives off the central retinal artery, which sends an anterior and posterior branch to the optic nerve." It would be conceivable, they state, "That the inflammatory process in this case had traveled in this direction."

Summary of the Literature: In practically all of these case reports the question of a sinusitis arose. In over 50 per cent, painful mastication at the onset is mentioned. This indicates that the inflammation of the temporal artery extended down or, in fact, may have originated at, or below, the temporomandibular joint region.

It is impossible to present accurate statistics at this time because, as has been mentioned, a number of cases have been observed by Horton and have not been reported as yet. But, in those reported and referred to, we find that the youngest patient was 55 years of age and the oldest 80. The average age was 67, and there were three times as many women as men, and all of the white race. The shortest duration was one month and the longest 20 months; the average was nine months. The age level and prolonged course with recovery are distinctive clinical features of a temporal arteritis.

AN ARTERITIS

In 1866, Kussmual and Maier introduced the term, "periarteritis nodosa." In this the clinical picture is that of a young adult around 30 years of age who has an acute or subacute inflammation of the smaller arteries, especially of the viscera. The diagnosis is difficult and is usually made at autopsy. As in temporal arteritis, the cause is not known and it is of special interest to us that in case reports of periarteritis nodosa a central retinal arteritis is often also mentioned.

What does the term, periarteritis nodosa, mean in reference to microscopic pathology? It means an inflammation of segments of an artery in which the tunica adventitia is primarily involved and a nodule is formed. This is true, but, although the tunica adventitia is primarily involved, the tunica media and tunica intima also show pathological changes, even to the extent that a thrombus may form in the lumen of the artery. Therefore, many pathologists prefer to designate it as a panarteritis. The microscopic pathology of temporal arteritis is essentially the same as in periarteritis nodosa; in fact, so much so that in Case 5 in my digest of case reports we find that "Dr. Gray, our pathologist, without knowledge of the case history, was unable to exclude periarteritis nodosa."

Horton and his associates[5] at the Mayo Clinic state "Their sections show they resemble in some respects periarteritis nodosa; the latter affects smaller vessels and tends to form aneurysmal sacs; and that giant cells have not been observed as in temporal arteritis." Although the microscopic pathology of temporal arteritis is quite similar to that of periarteritis nodosa, the clinical picture is entirely different.

Diagnosis and Treatment: In our two cases a diagnosis was made by the clinical picture in which a tender, red, tortuous, nodular temporal artery appeared. In the first case this occurred five weeks after the onset of the temporal pain, and in the second case, two

weeks. I wish to stress the point that this marked nodular swelling of the inflamed artery could be observed at a distance of 10 feet from the patient. I emphasize this because in each case (in the first at 12 months and in the second at 7 months) the artery was clinically normal in appearance and texture. Sulfonamides were tried and discontinued. We did not feel that a resection of a segment of the artery had therapeutic value. Our two patients made a complete and spontaneous recovery. However, one of these patients had a severe acute exacerbation of the temporal arteritis following extraction of three infected teeth. This episode would verify the opinion of some previous writers that a focus of infection is the causative factor. For example, Horton,[1] in presenting his second case report, calls attention to the fact that the patient recovered after extraction of all his infected teeth.

DIGEST OF CASE REPORTS

CASE 1.[1] Woman, aged 55.

February 1, 1931: "Onset of headache, soreness of scalp, painful nodules along the temporal arteries, anorexia and loss of weight."

March 16, 1931: "The patient entered the hospital and remained there 42 days. A few small painful nodules were still present in the left temporal region along the superficial temporal artery. They recurred from time to time during her stay in the hospital. The temperature ranged from 98° F. to 103° F. The white blood count varied from 7,500 to 13,700; the blood pressure varied from 140/60 to 190/100. The visual fields were normal."

June through August, 1931: "During this period painful nodules recurred along the temporal arteries with the usual systemic effects."

September, 1931: Local signs and general symptoms gone.

November, 1932: (13 months later) "Tender areas along the left temporal artery developed."

December, 3, 1932: The left temporal artery was more prominent and tortuous than the right, and small segments about 1 cm. in length were tender, but still pulsated. The blood pressure was 200/120. During this month signs and symptoms of temporal arteritis disappeared. (Temporal arteritis was present off and on for 20 months.)

February 27, 1933: The patient was admitted to hospital because of renal and cardiac failure. The visual fields were normal. She was in the hospital for 32 days, home for a month, and then died. No autopsy was performed. During her stay in the hospital a microscopic section was made of a segment of the temporal artery.

Comment: The section is like that found in periarteritis nodosa; a panarteritis "with marked thickening of the intima almost to the point of occluding the vessel. A granulomatous area with almost complete destruction of the media is present."

CASE 2.[1] Male, aged 68, had always had excellent health until:

April, 1931: "Onset of pain around the teeth and stiffness in the jaw. A week later, pain developed in the forehead and frontal areas. He developed painful and tender areas over the scalp."

May 25, 1931: Four weeks after the onset of pain, he was seen at the clinic. "He had lost 25 pounds and was very weak. His blood pressure was 165/90. The temperature ranged from 99° F. to 100° F.; leucocytes to 10,600. Red, raised, tender areas were present along the superficial temporal arteries. A large segment of the thrombosed left temporal artery was removed. Sections show a panarteritis and a thrombus in the lumen of the vessel."

Comment: These sections are similar to those found in articles on patients with periarteritis nodosa.

October 30, 1932: A letter from the patient reported that he had had no recurrence since the removal of all his teeth over a year ago.

CASE 3.[2] Woman, aged 60.

January, 1935: Onset with a tender area in front of the right ear. A week later the left side of her face became swollen and tender. At this time she became aware that the temporal arteries were prominent, red, and tender. Mastication was painful.

March 1, 1935: Five weeks after the onset she entered the hospital because of pain in both temples, fever, sweats, and general malaise. Her blood pressure was 148/90; temperature ranged from normal to 101.8° F.

March 29, 1935: Section of the right temporal artery was done after which the temperature became normal.

May 13, 1935: She returned to the hospital because the left temporal artery was inflamed. The eyegrounds were normal. Temperature ranged from normal to 99.6° F.; blood pressure was 116/86. Some teeth were extracted and she left the hospital on May 17, 1935.

August 6, 1935: Patient seen and she stated that she was well. The left temporal vessel appeared normal and there was a normal pulsation. No recurrences.

Microscopic sections of March show a panarteritis plus "massive hypertrophy of vessel wall and edema of connective tissue surrounding the arterial wall and inflammatory cell infiltration of this connective tissue."

Comment: This marked occlusive panarteritis and involvement of *adjacent* tissue is a characteristic of sections of thrombo-angiitis obliterans.

CASE 4.[3] Woman, aged 68.

June, 1936: "Onset with earache, pain in the jaws, back of the head and in the eyes, and weakness. These symptoms persisted for four weeks with no clue as to the basis for the complaints until the temporal arteries became markedly inflamed, thickened, and tortuous."

Microscopic section of the temporal artery shows: "Occluded vessel; the lumen is filled with a recent thrombus. All three coats are markedly thickened."

Comment: The writers state that "a study of their sections show they resemble, in some respects, periarteritis nodosa—the latter affects smaller vessels and tends to form aneurysmal sacs; giant cells have not been observed as in temporal arteritis."

CASE 5.[4] Woman, aged 66.

May, 1936: Onset with extreme fatigue; the next month she had pains in the thighs, knees, and ankles which became so severe that she was confined to bed. In August all extremities were painful and she had fever and sweats. By November the pains decreased; she went out, but was unable to walk. In December she had stiffness in the neck.

January, 1937: There were generalized headaches, her mouth became stiff and her ears felt swollen. The back of the head was tender and she was able to feel tender little swellings on her forehead.

February 2, 1937: A spot appeared in front of the left eye and during the next five days "it was as though a curtain was being drawn over the left eye, removing all sight from it."

February 8, 1937: On admission to the hospital, haziness began to develop in the right eye. She had a marked photophobia. The blood pressure was 160 90. The left eye was found completely blind, with a large pupil and no light reflex. The disc was pale, there was a small central hemorrhage, and the retinal arteries were narrow. The picture was that of definite obstruction of the central artery. Both temporal arteries, especially the left, were tortuous, red, and tender, and pulsations in each diminished.

February 17, 1937: The right eye had rapidly deteriorated. The disc was pale; there was a small hemorrhage below and near the disc; no vision in the lower half of the field.

March, 1937: The inflammation subsided and the temporal arteries were not tender. Headaches, eye pains, and photophobia had disappeared. The left eye remained blind. There was no increase in the field of vision in the right eye, but she was able to read large print. She was in the hospital six months, had no fever, and a left radial arteritis was noted.

March 10, 1937: During her recovery a segment of the right temporal artery was sectioned. In August 1937, she left the hospital and in December was reported well except for her eyes and that the left radial artery could not be felt.

Comment: They state: "It is significant that Dr. Gray, our pathologist, without knowledge of the case history, found an appearance resembling rheumatic arteritis and at the same time was unable to exclude periarteritis nodosa."

Conclusion: The patient had a temporal, retinal, and radial arteritis.

CASE 6.[1] Woman, aged 72, a patient of Coles of Cambridge, England, who reported to the writers.

January, 1931: "She began to feel weak and out of sorts; her appetite was bad and then she developed a pain and tenderness in the right temporal region which was worse at night. After a few weeks the same symptoms developed on the left side and were more severe. She thought she had a slight fever; otherwise, there were no other symptoms."

On Admission: (No date) "Woman looked ill. Marked arteriosclerosis present. Temporal arteries on each side were thickened, red, tortuous, painful, and tender. Temperature ranged to 100° F.

Comment: Coles states, "that the diagnosis made then was subacute periarteritis, but the condition was very strange."

CASE 7.[5] Woman, aged 71.

December 2, 1937: Onset with pain in the neck and temperature of 102° F. Two days later she had pain in the face and red, tender nodules in each temporal artery. She gradually improved but on

December 16, 1937, she became disorientated, complained of pain in her chest and could not move her arms to feed herself. The blood pressure was 136/80; temperature, 100° F.; white blood count, 12,500. Nodules in temporal arteries were still present.

December 30, 1937: Gradual and complete improvement and nodules disappeared.

CASE 8.[6] Woman, aged 65.

May, 1938: The patient suffered from bronchopneumonia lasting five weeks, after which the right side of the face became tender and swollen and a week later the left side became similarly affected. The pain was prominent in the jaws and mouth. Cord-like swellings were felt along the occiput, behind the ears, and over the temples.

Entered Hospital: Patient was acutely ill and undernourished. The above findings noted. The retinal vessels appeared engorged. The radial and brachial arteries were thick. The temperature ranged to 100° F; white blood count to 13,000. While the patient was in the hospital, the vision in the left eye became blurred. The optic disc was twice the normal size and slightly edematous. A small hemorrhage was present on the disc. The visual field was reduced to a small point.

September, 1938: Patient was well. A section of the right temporal artery removed while she was in the hospital shows a panarteritis, and attention is called to the giant cells in the media.

CASE 9.[6] Woman, aged 76.

November, 1937: She began to have severe, persistent, frontal headaches and aching in the face, eyes, and jaws.

April, 1938: Tender swelling of the left temporal artery appeared and then the same on the right side. Soreness of the eyes, blurred vision, and diplopia appeared.

June, 1938: Examinations made which revealed large, firm, tortuous, pulsating temporal arteries, tenderness of the scalp, sluggish extra-ocular movements, and poor vision in the right eye. The blood pressure was 172/80; the temperature ranged to 100.4° F.

June to October, 1938: The headaches were absent, but the patient did not improve.

December, 1938: Vascular signs subsided. (Vision in the right eye at this time is not mentioned.)

CASE 10.[7] Woman, aged 65. Onset with occipital pains which lasted five weeks, then temporal pains which lasted six weeks. These pains had been constant, dull, throbbing, and worse at night, and so severe that they caused malnutrition.

September 20, 1937: (In hospital.) The temperature ranged to 101.4° F.; the blood pressure was 160/110. Both anterior branches of the temporal arteries, and especially the left, were thickened, tortuous, and without pulsation, but were not nodular. The occipital arteries were still tubular and thick walled, not painful, but very tender. The pain in the temples was excruciating, especially on the left.

September 27, 1937: Segment of left temporal artery removed. "The patient in a short time commented on her delight in the relief from severe pain at the site of removal of the biopsy specimen; presumably this relief was due to interruption of the pain pathway of the sympathetic nervous system."

October 17, 1937: The patient left the hospital, though weak. Blood pressure 138/94.

January 19, 1938: Due to prolonged worry, the blood pressure was 220/110, but she had no pain in the occipital or temporal arteries.

September, 1938: (One year after she was first seen.) She again appeared in the office, hardly able to walk or talk because of profound weakness. Blood pressure 230/120. No localized inflammation of the peripheral arteries was present. She was re-admitted to the hospital because of vomiting and exhaustion, and she sank into a fairly deep coma which lasted five days. Supportive measures were used and in six weeks the patient left the hospital.

Comment: The author states, "The same form of arteritis that afflicted the temporal vessels may have involved the cerebral vessels in September, 1938. That is, 'an arteritis of the cerebral vessels.' "

Biopsy Section: This shows a panarteritis and thrombus in the lumen of the temporal artery.

CASE 11.[8] A man, aged 66, was in good health until

October, 1939: Onset of pain in left side of the face, simulating an atypical temporal neuralgia.

November 21, 1939: The patient was first seen; was poorly nourished. Blood pressure 150/85.

December 2, 1939: The pain had shifted to the right side of the face, and especially the temporomandibular joint. The right temporal artery was prominent and tender. The white blood count was 12,900.

December 4, 1939: The left temporal artery was prominent and tender. A biopsy of the temporal artery was made.

December 17, 1939: Because his condition became worse, the pain was severe, and because of increasing anorexia, he became dehydrated and was sent to the hospital where he was given daily administration of glucose in saline for one week. Opiates were given for the pain. Low-grade sepsis present.

December 24, 1939: The patient was sent home after being advised of diagnosis and slow prolonged course. Mentally he was very sluggish, like an encephalitic.

March 7, 1940: The inflammatory reaction in the arteries had subsided considerably. He was only able to be up part of each day.

April 23, 1940: Gradual improvement, but the patient was still weak and mental alertness had not returned.

Comment: Biopsy of temporal artery shows a panarteritis. The writers state, "The possibility of permanent cerebral involvement (cerebral arteritis) is suggested in this case."

CASE 12.[9] Woman, aged 70.

December, 1939: Onset with weakness and loss of appetite. A few weeks later she had burning pains in the arms and hands in the course of the large arteries.

January, 1940: Pains in the arms subsided, but were followed by similar pains in the neck and the occipital regions, and about both ears.

March, 1940: "Scalp sore and tender, and reddened, painful nodules appeared over both temporal and frontal regions." *Note*: Case report states that, "During the entire course of her illness she had no appetite and had severe night sweats on numerous occasions."

April 2, 1940: The patient entered the hospital. "The temporal arteries were tortuous, thickened, tender, and there was some induration of the surrounding tissues. There was tenderness over the facial artery anterior to each ear, and the carotids were tender. Eyegrounds were normal. Blood pressure 110/70."

April 8, 1940: Segment of right temporal artery excised for study. *Note*: "Complete and immediate relief of symptoms over the site operated upon; hence, the left side was resected four days later, with definite, but not as complete relief of pain on that side."

Further Course: "In spite of this relief, her constitutional symptoms persisted and she continued to have a temperature to 100.4° F.".

June, 1940: "She returned with the complaint of loss of vision in the right eye and some diminution in the left. Her head pains were slight, but she gained no appetite nor strength. The right fundus could not be seen, due to lenticular opacity. The retinal arteries of the left eye had small patches of exudate."

Final Course: Patient lived, but no more mention is made of the eye findings or whether there had been an improvement of her constitutional symptoms.

Biopsy Section: This shows a panarteritis in which attention is called to the giant cells in the media.

CASE 13.[10] Woman, aged 62. Onset with severe right temporal pain, fever, sweats, and general malaise; followed by dizziness, gastric distress, and vomiting. Because of the grave condition of the patient and the severity of her symptoms she was

Admitted to Hospital: (No date given) where examination revealed that "the right temporal artery was prominent, tortuous, thickened and tender, and pulsation was absent. Blood pressure 155/80. Temperature ranged to 101° F. All tests were negative. The patient's condition became progressively worse. The headache

could not be controlled by one-half grain of codeine. She appeared desperately ill, her speech became blurred, and finally she became delirious. The question of a brain tumor with increased intracranial pressure was considered, but ruled out by a neurologist. The patient did not obtain any relief until two grains of sodium luminal were given intramuscularly. On awakening, the symptoms were as severe as ever."

The Cure: "Finally the patient was put on the Sanders oscillating bed and enjoyed her first symptomless and natural sleep without any medication." This oscillating bed was used in the hospital for ten days and then in her home for six weeks. "She made a remarkable recovery and is entirely free from symptoms."

December 2, 1941: "None of the symptoms have returned; the final diagnosis of temporal arteritis was made from the clinical manifestations."

Comment: It is obvious that the patient had an arteritis of the temporal artery. But, the major involvement, as judged by the history, is a cerebral arteritis. A localized temporal arteritis could not have produced such a profound clinical picture of cerebral reactions. The relief and cure by the Sanders oscillating bed is very dramatic, and, as the writers state, "The result exceeded our expectations."

CASE 14.[11] Woman, aged 71. Onset with severe occipital pain, swelling of the left jaw, night sweats, and a painful swelling of the vein of the head.

Examination three days later revealed a bright, well nourished woman who did not appear acutely ill. Along the course of both superficial temporal arteries an area of erythema was observed and palpation revealed firm, nodular cords which were tender, especially so on the left. The area over the muscles of mastication on the left side was also tender. Blood pressure 190/110.

17 Days Later: A segment of the left temporal artery was removed. *Note*: "The pain rapidly subsided after this procedure, but mild complaints of exhaustion and vertigo persisted for six months."

9 Months Later: Patient very well. Section for pathological study shows a typical panarteritis.

CASE 15.[12] Woman, aged 61.

June, 1938: "Onset with fatigue and anorexia. These symptoms became progressively more marked."

August, 1938: "Sickening pain about the forehead; this pain extended toward the temples, was intermittent, and became progressively more severe and associated with nausea and vomiting lasting five days. Then the vessels about her temples were observed to be swollen and tender. She described the temporal pain at this time as severe, paroxysmal, lacrimating, and extending about the eyes and cheeks. Between the intervals of pain she would feel well but exhausted."

December 6, 1938: The patient entered the hospital because of visual disturbances which started two weeks previously. At first the right eye became involved. This arterial retinitis subsided, but the one in the left eye produced total loss of vision. The patient recovered but had no vision in the left eye.

Biopsy of the left temporal artery demonstrates a panarteritis.

CASE 16.[12] Man, aged 75.

February, 1942: Onset of dull, constant, boring, left, frontotemporal pain. A week later, tender nodules appeared on the left temporal region.

March 2, 1942: There was sudden loss of vision of left eye.

March 5, 1942: Onset of right temporal arteritis; "several small nodules had appeared on the anterior portion of the temporal region. These nodules were tender and their appearance had been preceded by right frontal headache. The vision of the right eye was limited by a dense leukoma to perception of the movements of the hand. Vision in the left eye was completely gone."

March 9, 1942: "On the evening of the day of registration to the hospital, exacerbation of the right temporal pain was accompanied by sudden, complete loss of vision in the right eye."

CASE 17.[12] A woman, aged 76, was under observation with blindness in the right eye, with bilateral temporal arteritis, more marked on the right side.

AUTHOR'S CASES

CASE 18. Woman, aged 76.

January 6, 1941: Onset of pain in the right temporal region, malaise, leucocytosis, and a temperature of 100° F. The temporal pains were decidedly worse at night. Because of some nasal congestion, the question of a sinusitis arose. The temporal arteries appeared normal. No relief was obtained by nasal treatments. In fact, even

after the nasal congestion had subsided the temporal pains became so severe and penetrating that the patient was sent to the hospital on

January 28, 1941: (three weeks after the onset) for observation, and remained there a week. The patient had lost 15 pounds, had a white count of 14,500, and a temperature which varied from normal to 100° F. Her blood pressure was 150/70. A cerebral involvement was considered and ruled out by x-ray and neurological examinations by Dr. Ussher. The eyegrounds were normal.

February 10, 1941: (5 weeks after the onset) The pain had become localized to the right temporal artery which now, for the first time, was red, prominent, tortuous, and nodular; pulsation was present.

February 23, 1941: The symptoms had subsided a great deal. The temporal artery was less inflamed and she had no fever.

February 28, 1941: Extraction of three infected teeth was immediately followed by a severe, acute exacerbation of the temporal arteritis. The artery was extremely tender and painful, and now pulseless.

March through December, 1941: The patient was in bed most of the time because of malaise, temperature, and temporal pain. The recovery was slow, but complete. The treatment was mainly empirical and supportive.

January 8, 1942: (One year after the onset) The temporal artery had a normal pulsation and texture.

January 10, 1944: A personal interview with this woman reveals that she has had no recurrence of the temporal arteritis and is very well. The temporal arteries are normal and, in fact, show no signs even of arteriosclerosis, although she is now almost 80 years of age.

Comment: In this case one would infer that the severe, acute exacerbation of the temporal arteritis immediately following the dental extraction would suggest a focus of infection as the causative factor. The ultimate complete restoration of normal function and appearance of the artery is significant.

CASE 19. Woman, aged 61, partially incapacitated by virtue of angina pectoris and hypertension of long standing.

June 23, 1941: Onset with severe pains in both temporal regions which extended down to the mandibular joints. Mastication

of food, therefore, was painful and difficult. These pains were immediately associated with generalized headaches, nausea, malaise, prostration, and fever. Physical examination and many laboratory tests by Dr. Elliot failed to disclose the cause of her difficulties. Because of the temporal pains, headaches, and fever, a question of nasal sinusitis arose. It must be admitted that at this early stage, with this history and no diagnosis possible, a sinusitis had to be considered. Nasal examinations and local nasal therapeutic procedures failed to reveal a sinusitis or relieve the temporal pains or headaches.

July 7, 1941: (Two weeks after the onset) The patient for the first time complained of tender spots on her scalp. Inspection disclosed an involvement of both anterior temporal arteries. Although pulsating, they were tortuous, indurated, nodular, and very tender. At this date (July 7, 1941) the patient was hospitalized for one week. During this week the temperature ranged from normal to 100° F.; the white blood count from normal to 12,000. Three grains of nembutal were given every night, with 2 gr. of codeine, and 6.0 gm. of sulfathiazole every 24 hours.

July 10, 1941: Photograph taken of red, tortuous, nodular, left temporal artery, now pulseless.

July 14, 1941: The patient was sent home after being informed of the diagnosis of temporal arteritis and anticipated prolonged course and recovery.

August 17, 1941: (6 weeks later) She felt stronger; the pain was decreasing. Both temporal arteries, although not tender, were nodular and pulseless.

October 17, 1941: (2 years later) The general vascular disease had not progressed. The temporal arteries were normal. No visual disturbances occurred during or following her temporal arteritis.

Comment: In the early stages of the inflammation the artery was nodular and had pulsation. Later the pulsation disappeared due to an occlusion of the lumen of the artery, but reappeared in the reparative stage. Eventually the artery also resumed its normal appearance and texture, as in our first case.

CONCLUSIONS

The conclusions are based upon the observation of my two cases and a careful scrutiny and digest of all the case reports in the literature.

1. A rhinologist should incorporate this subject in his differential diagnosis because he may be called in to see the patient in the early stages before a final diagnosis is possible. The temporal pain, fever, leucocytosis, malaise, and sweats are present two to six weeks before the temporal artery becomes red and prominent.

2. The microscopic pathology is essentially the same as in periarteritis nodosa, but the clinical picture is entirely different.

3. The high incidence of an associated retinal arteritis in which the temporal arteritis subsides, but permanent decrease or even loss of vision occurs, must be borne in mind.

4. We present two case reports. In these we emphasize the fact that although the temporal artery was red, nodular, tortuous, prominent, and pulseless at the height of the involvement, at recovery it had a normal appearance, texture, and pulsation.

5. The severe, acute exacerbation of the temporal arteritis immediately following the extraction of three infected teeth and ultimate recovery in our first case would suggest a focus of infection as the causative factor. This verifies the observations of some other writers, but does not explain the etiology in the majority of the case reports in the literature.

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Clinical Notes

XXXI

NEUROFIBROMA OF THE PHARYNX WITH PARALYSIS OF THE LARYNX FOLLOWING OPERATION

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Neurofibroma of the pharynx must be considered as occurring infrequently as the literature contains few reports of such cases. Figi,[1] in his report in 1933, quoted Penfield as suggesting the term "perineural fibroblastoma" for tumors variously classified as solitary neurofibroma, neurinoma, fibroma of nerves, gliofibrosarcoma, peripheral glioma, schwannoma and acoustic neuroma. Cases have been reported by Motley,[2] and by Hall and Owens,[3] both in 1941, and by Foster[4] in 1942.

This case is of interest because of the occurrence of a postoperative laryngeal paralysis.

A. P., a young woman of 20, was seen because of obstruction and feeling of fullness in the left side of the throat. She had had a tonsillectomy eight years before. Just recently she had been admitted to another hospital with a diagnosis of peritonsillar abscess.

Examination disclosed a marked bulging of the left lateral wall of the pharynx extending from the level of the base of the tongue, upward behind the soft palate, almost to the torus tubae. This was rather firm to palpation. Laboratory studies and x-ray films of the neck and the nasopharynx were negative. The pre-operative diagnosis was fibroma or mixed tumor.

Under ether anesthesia the mass was exposed by a longitudinal incision and a large encapsulated tumor, extending nearly to the base of the skull, removed by blunt dissection. This was about the size and shape of a hen's egg, measuring 6 cm. by 4 cm., and was of yel-

Read before the Sixty-Sixth Annual Meeting of the American Laryngological Association, New York, N. Y., June 8, 1944.

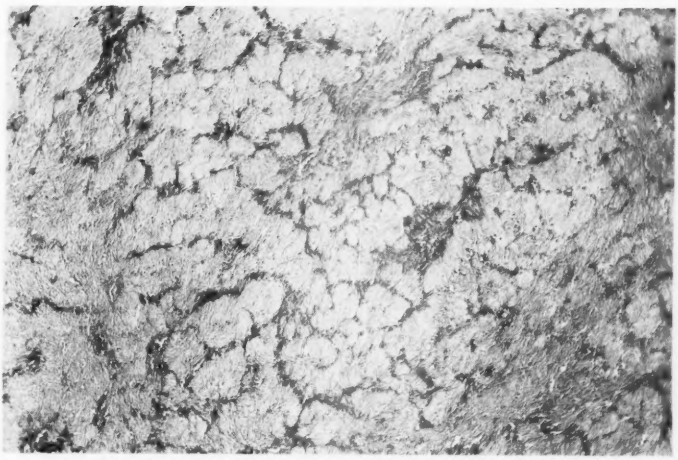

Fig. 1.—Photomicrograph of section of tumor; x70. The cells are arranged in regimental formation or palisades, typical of the cells of the neurilemma or Schwann sheaths of the fibers.

lowish-white color. The incision was closed with silk, and healed promptly.

The histopathological examination by Dr. J. Gottlieb, showed, "Neurofibroma or neurinoma, resembling an acoustic nerve tumor. Can be considered as a pure Schwann cell tumor."

Drooping of the left eyelid and a contracted pupil, indicating involvement of the cervical sympathetics, were noted on the second postoperative day. These subsided in two days.

Of more significance was the finding of a left abductor laryngeal paralysis. The patient complained of a weak voice and examination of the previously normal larynx now disclosed the vocal cord motionless in the midline position. I felt that this might be a temporary condition, possibly due to pressure or hemorrhage, but the condition has persisted now for a period of eight months, the only change being a shift of the cord to the paramedian, or cadaveric position. There is no anesthesia of the larynx. She is otherwise perfectly well and has no complaints other than a rather weak voice.

COMMENT

The findings suggestive of Horner's syndrome, similar to those noted in Foster's case, were due probably to the postoperative reaction, as they promptly subsided. The postoperative development of laryngeal paralysis suggests the vagus nerve origin of this tumor, probably adjacent to the jugular foramen, as palpation of the postoperative cavity revealed it to extend nearly to the base of the skull. Only a portion of the efferent fibers were involved as there was no paralysis of the soft palate or the pharynx, nor any anesthesia of the larynx. The first finding of the vocal cord in adduction, with a later change to the cadaveric position, suggests progressive involvement, first of the more susceptible abductor fibers, followed later by the adductors.

Possibly operation by means of an external approach would have resulted in less trauma to the vagus nerve. While the intraoral procedure was not especially difficult, it was necessary to exert some traction by applying a tenaculum to the tumor. However, the origin of the tumor was not established until after operation, and with the thought that it was a mixed tumor or some simple fibroma, there was a natural inclination to avoid an external scar.

The present laryngeal picture is typical of the usual recurrent laryngeal nerve paralysis, with the vocal cord in the paramedian position and the arytenoid slumped forward. It suggests the advisability of keeping in mind that such findings may, at times, be due to pathological conditions at the base of the skull, as well as in the neck of the mediastinum.

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XXXII

ATRESIA OF THE PHARYNX OPERATED UPON BY THE MACKENTY METHOD

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J. A., male, aged 3½ years, was first seen in October, 1943.

The chief symptoms were some slight difficulty in swallowing, noisy breathing, inability to breathe through his nose and a muffled voice. All these symptoms followed within a few days an operation intended to remove this child's tonsils and adenoids. Since last June, he has neither gained in weight nor stature.

Physical Examination. This child's weight was 30 lbs. (13.5 Kg.); his height, 40 in. (1.016 meters).

The nasal examination revealed that on inspiration the alae dilate, while on expiration there is no expiratory blast. A very small amount of mucoid discharge was present in the inferior meatus, both sides.

The teeth, gums and tongue were normal. There was a normal amount of saliva in the mouth.

The oropharynx was not visible, for there was a web of scar tissue that stretched from the hard palate, including the soft palate and the uvula, in a downward direction to the posterior wall of the lower part of the oropharynx, approximately at the level of the dorsum of the tongue, when depressed. No landmarks could be identified in this web of tissue. At the lower part near the junction with the pharyngeal wall, there was a small fistula into which a fine probe could be inserted upwards for approximately 1 cm. When the sutures were in place and traction made, the outline of the uvula could be seen (Figs. 1 and 2).

The ear drums were considerably thickened.

Examination by direct laryngoscopy showed that the epiglottis and the laryngeal structures were normal. The Wassermann test was negative.

Read before the Sixty-Sixth Annual Meeting of the American Laryngological Association, New York, N. Y., June 8, 1944.

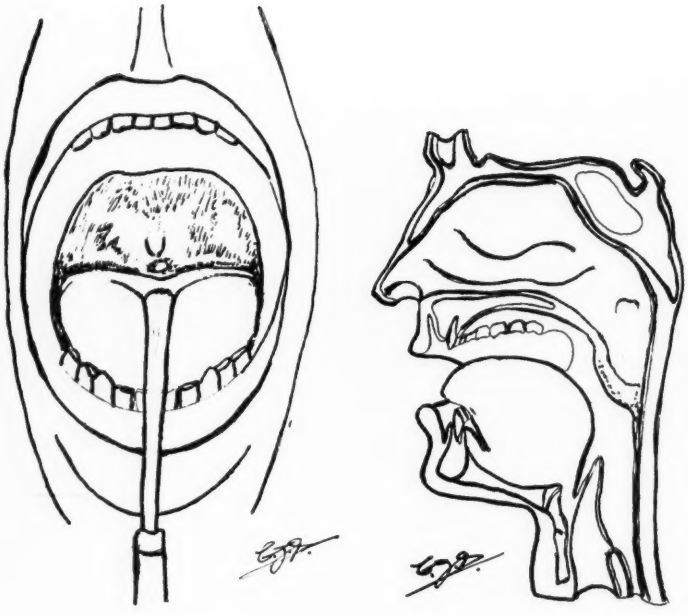

Fig. 1.—This illustration shows the scar tissue stretched across the oropharynx. The uvula can be just outlined imbedded in it and just below it is a small fistula, that, on probing, leads in an upward direction. Because of the irregular course, it did not permit of admittance of the probe into the nasopharynx. However, occasionally, an air bubble could be seen in the opening of the fistula.

Fig. 2.—Lateral view (diagrammatic) of the atresia in relation to the other structures.

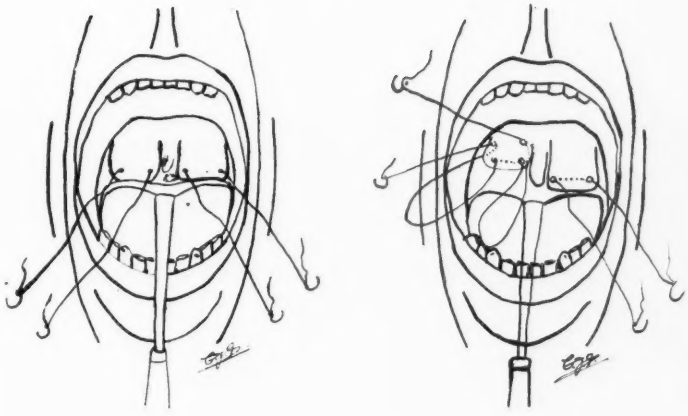

Fig. 3.—Suture retractors are placed in the lower part of the atresia just above its attachment to the lower pharyngeal wall. The suture material should be of a material similar to silkworm gut. In placing them, pass the needle, preferably a No. 2 curved, in a needle holder that will hold the needle at an angle of 45 degrees. Pass the needle through the atresia from within outward and into the depth of the atresia sufficiently that the posterior surface is included. The sutures should emerge near the posterior pillar or what was that anatomical entity.

The suture is then armed with a needle of the same size just used in its proximal end.

This procedure is repeated on the left side and the "U" shaped flaps cut, as illustrated.

The sutures are used as retractors and the adhesions are separated, preferably by sharp dissection.

Fig. 4.—The sutures are now introduced from behind the soft palate and the flaps drawn into the nasopharynx and anchored to the nasopharyngeal surface of the soft palate.

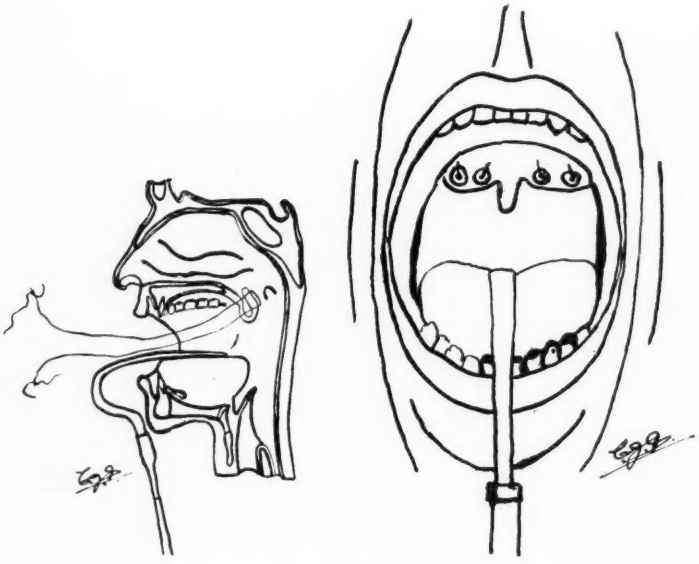

Fig. 5.—This will illustrate a lateral view of a flap drawn up.

Fig. 6.—Completed operation with the sutures anchored with split shot. (It is important not to draw the sutures too tightly or the shot will produce a pressure necrosis.)

Diagnosis: Pharyngeal obstruction produced by a web of scar tissue closing off the oro- and nasopharynx.

Operation: On November 16, 1943, an operation was performed following the procedure as described by Mackenty[1] and Wright and Smith.[2]

In dissecting the flaps, as described by Mackenty, the uvula and most of both tonsils were found imbedded in the scar tissue. There was a very prominent projection from the body of the second cervical vertebra to which most of the scar was attached posteriorly (Figs. 3-6).

The present condition (May 4, 1944) of this child is very satisfactory. His weight is 35½ lbs. (16.10 Kg.); his height, 41.5 in. (1.054 meters).

In order to keep the opening from inverting, particularly on one side, it was necessary at the weekly and later monthly examinations, to dilate the opening. This was done with a Kelly clamp, the ends of which were covered with rubber tubing.

The nasopharyngeal opening is approximately two-thirds that at the time of the completed operation. The voice is good, the breathing appears to be normal and the expiratory blast from the nostrils appears to be normal, so that there is no difficulty in blowing his nose.

The type of operation that was done by the physician is not known but it is assumed that the injury to the tissues was probably produced by the use of a curette. Formerly, these injuries were common. Undoubtedly, those individuals, having a prominent tubercle on the atlas or a very prominent body of the axis, were more likely to have this sequela and particularly so when the curette, such as the Gottstein or its modification, the Beckman, was used. Since the introducton of the LaForce adenotome, these sequelae are infrequent.

In examining the reports of the Manhattan Eye, Ear and Throat Hospital, where approximately 100,000 adenoid and tonsil operations have been done in the past ten years, there are but three cases recorded of atresia of the pharynx. Two of these patients were adults and one a young child; two followed an adenoid and tonsil operation and the other followed a severe throat infection. No details as to the type of tonsil operation are given and no note as to the Wassermann reaction is recorded. In two patients, the

Seton type of operation was done, and on the other no details of the operative procedure were given. There is no record of the final results.

One would conclude that this sequela following an adenoid and tonsil operation is comparatively rare, when the accepted technique is practiced.

108 East 38th Street.

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2. Wright, Jonathan, and Smith, Harmon: Diseases of the Nose and Throat. Philadelphia, Lea and Febiger, 1914, p. 651.

XXXIII

CANCER OF THE LARYNX
REPORT OF UNUSUAL CASE

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Philadelphia, Pa.

The expectancy of life of a patient with untreated carcinoma of the larynx is dependent upon a number of factors. For that reason it is hazardous to express an opinion regarding the probable length of life. Few untreated patients survive for more than two or three years after a diagnosis has been established.

The following case is of interest because of the original diagnosis and therapeutic recommendations, the subsequent course and duration of symptoms and the final outcome. The following history was secured from the patient's son, a physician.

Male, aged 77 years, consulted me at the Jefferson Hospital during October 1938. There was a history of hoarseness of over 11 years' duration with dyspnea of about one year's duration. During early 1927 the patient developed hoarseness and was referred to the late Dr. J. E. Mackenty by his physician during the latter part of October of the same year. Microscopic study of tissue removed from a tumor of a vocal cord was diagnosed as "an invasive form of epithelial new growth." Dr. Mackenty recommended laryngectomy but the patient refused this and returned home. He continued with his activities and was in charge of a general store for approximately ten years when he began to have dyspnea and was no longer able to carry on his work. Recently the dyspnea became marked and he finally decided to have some plan of surgical treatment carried out.

On examination the patient's general condition seemed excellent for one of 77 years. The outstanding physical findings were limited to the larynx. There was an extensive ulcerated lesion which involved the entire left vocal cord, extended subglottically for a considerable distance and invaded the anterior extremity of the opposite vocal cord. The left side of the larynx was fixed. The right

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From the Department of Laryngology and Broncho-Esophagology, Jefferson Hospital, Philadelphia, Pa.

Read before the Sixty-Sixth Annual Meeting of the American Laryngological Association, New York, N. Y., June 8, 1944.

Fig. 1.—Photomicrograph (X83) from biopsy specimen exhibited a bit of mucosa and solid clumps of epithelial cells which are infiltrating. The epithelium is fairly well differentiated and there is formation of keratin (Dr. B. L. Crawford).

vocal cord and subglottic tissues appeared edematous but there was no impairment of motility of that side. There was no demonstrable evidence of metastasis. Roentgenological examination of the chest revealed no evidence of pulmonary metastasis and systemic studies all were essentially negative.

A biopsy was done and additional tissue was removed to improve the laryngeal airway as there was marked stenosis. The histologic diagnosis by Dr. Baxter L. Crawford was papillary squamous-cell carcinoma, intermediate grade of malignancy (Fig. 1).

The question of therapy was discussed with the patient and his son. In view of the need of tracheotomy for dyspnea and the absence of any demonstrable metastasis to regional lymph nodes it was decided to do a laryngectomy. Because of subglottic involvement it was deemed advisable to remove the first and second tracheal rings; in addition, a large segment of hyoid bone was removed.

The larynx was divided in the midline posteriorly following removal and the interior inspected. The growth appeared as an extensive ulcerating papillary lesion. It involved the entire left vocal cord extending into the ventricle and involving the anterior extrem-

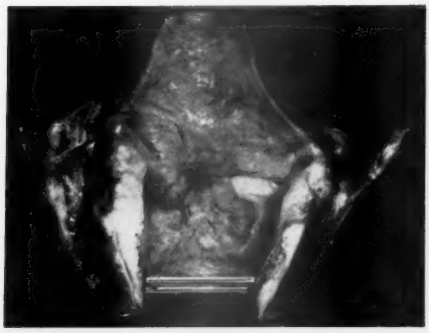

Fig. 2.—Specimen of larynx divided in midline posteriorly exhibited extensive involvement of the entire left vocal cord extending subglottically to the lower border of the cricoid cartilage, also involving the ventricle and anterior extremity of the left ventricular band and crossing the midline anteriorly involving the anterior third of the right vocal cord and corresponding subglottic tissues.

ity of the left ventricular band. It also extended across the midline involving the anterior third of the right vocal cord. There was marked subglottic extension below both cords, particularly anteriorly and on the left side where it extended to the lower border of the cricoid cartilage (Fig. 2).

The patient made a satisfactory postoperative recovery and returned home 24 days following operation. He has continued in excellent condition and there has been no evidence of recurrence now over 5½ years following operation.

COMMENT

The long interval from the time of the occurrence of symptoms and the original diagnostic studies in 1927 when laryngectomy was recommended and the time that it actually was performed, the comparative freedom from serious symptoms for a period of 10 years in the absence of any therapy with the later development of progressive dyspnea and increasing hoarseness, and the fact that laryngectomy still could be successfully performed 11 years after the original diagnosis was made immediately raise the question of diagnosis.

Should isolated cases of carcinoma which exhibit unusual behaviour be regarded with suspicion and should the pathological ma-

terial be reexamined in the light of these changes? When the diagnosis of carcinoma was made in 1938 a communication was sent to the pathologist who made the original diagnosis in 1927 and the histologic material was reviewed. There apparently was no reason for changing the diagnosis and this was considered an unusual case. There is no question that the larynx exhibited an extensive cancer when removed in 1938. A more recent review of the original biopsy material by another pathologist suggested that the condition might be considered as a case of Bowen's disease.

Bowen's disease is regarded by many pathologists as a highly specialized form of superficial squamous-cell epithelioma with lateral intra-epithelial spread. It more often is observed in the skin than in the mucous membranes. Its transition into frank cancer more often occurs in mucosal than in cutaneous cases. Brighton and Altmann's case is the first reported instance of a laryngeal localization.

1530 Locust Street.

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XXXIV

BILATERAL EIGHTH NERVE PARALYSIS FOLLOWING APPENDECTOMY UNDER AVERTIN GAS-OXYGEN-ETHER ANESTHESIA

REPORT OF A CASE

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This is a case report of a twelve-year-old girl who developed a bilateral eighth nerve paralysis following an appendectomy in which avertin and nitrous oxide-oxygen-ether were used as the anesthetic. A recent search of the literature has failed to disclose a case of similar nature. No specific cause is offered for this neurological accident. Correspondence from the manufacturers of avertin states that they can find no published descriptions of a similar accident in which avertin was used as the basal anesthetic agent.

Courville[1] believes, "that nitrous oxide produces its anesthetic effect, in part at least, by its narcotic action on the nerve cells of the cortex, and possibly the basal ganglia as well." Lundsgaard and Van Slyke[2] state that, "it is now well known that cyanosis is not synonymous with anoxemia and that anoxemia may occur without it."

It is also well known that cerebral complications following surgical procedures in which nitrous oxide-oxygen-ether was used may result in cerebral anoxia and be followed by cerebral complications. A few of the reported complications are amaurosis, difficult speech, residual athetosis, emotional imbalance, hemiparesis, aphasia and apraxia. Behrend and Riggs[3] have stated, "that the anoxemia causes cellular degeneration throughout the brain, generalized capillary damage with widespread parenchymal degeneration due to pericellular and interstitial edema." In the cases where decreased or complete loss of vision by injury to the cortex was recorded, Courville[1] has stated, "that the other centers of the cortex for general or special sensation seem not to suffer specifically as a result of anoxemia."

Read before the New England Otological and Laryngological Society, Feb. 16, 1944.

It would appear that those who eventually recover from neurological damage following anoxia usually do so within twenty-four hours, although at times it may take days or weeks to effect a slow and progressive recovery. Mental and emotional symptoms are found most commonly, and some writers feel that they are present to some degree in every patient. The case under observation had none of these symptoms to our knowledge.

It is also an established fact that avertin in many instances is a respiratory depressant and may cause a marked fall in blood pressure. There was no apparent cause found to contribute to the untoward effects in the pre-operative medication the child received and no indications of the part this may have played in any respiratory depression. In cases as severe as the one here presented, it has been noted that either during or following the operation one of the following symptoms is usually present: convulsions, apnea, lowered blood pressure, or disturbed respiration. This patient, according to the history, presented none of these symptoms. As the case came under observation five months following the operative procedure, only the history and findings as described on the initial clinical record and those elicited from the mother, along with the findings at the present time, are offered. It is worthy of mention that at the patient's first visit it was felt she might be suffering from hysterical paralysis. However, as the examination progressed and a complete, bilateral vestibular loss was found, this opinion was abandoned.

REPORT OF A CASE

This twelve-year-old, Irish-American girl weighed 35.5 kg. at the time of her hospital admission. According to the mother, she developed rather sudden pain in her abdomen on May 10, 1943, while the mother was at work. On the mother's return a pediatrician was called and a diagnosis of acute appendicitis was made. The child was sent to the hospital. There was no history of nausea, vomiting, constipation or diarrhea. The child had a temperature of 100° F. and had a white blood count of 12,000 on admission; no differential count was made. The surgeon noted that the abdomen was spastic.

Operation was deferred until the following morning, at which time the white blood count had risen to 19,000. As pre-operative medication the child was given seconal gr. .75 at 5:30 A. M., and at 7:15 A. M. she was given morphine sulfate gr. 1/12 and hyoscine hydrobromide gr. 1/300. At 7:50 A. M. she was given a 30 mg. per kilo basal dose of avertin over a twenty-minute period. Operation was begun at 8:15 A. M. and finished at 8:40 A. M.

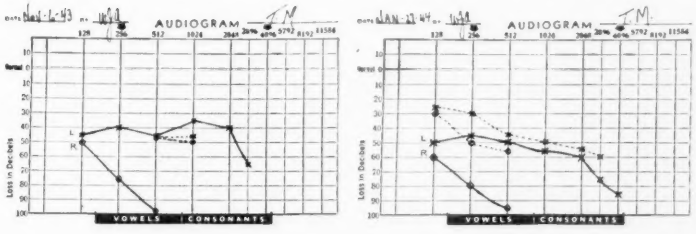

Fig. 1.—Audiogram Six Months Following the Operation.

Fig. 2.—Audiogram Eight Months Following the Operation.

The following notes are taken from the anesthetic record: no excitement; no vomiting; respirations, quiet; color, flushed; pulse, good quality; relaxation, good; complications, none; mucus, none; shock, none; condition after operation, satisfactory.

The blood pressure ranged from 105 to 115 systolic during the thirty minute operative period. Respirations were from twenty to thirty per minute. There was an apparently normal return to consciousness and at 3:30 that afternoon the child was given morphine sulfate gr. 1/12 for restlessness. The inability to hear was charted in the nurse's notes two days following the operation. The child's temperature dropped to normal the day following operation and from 99° F. on the following day it gradually returned to normal. She left the hospital on May 22, 1943.

Extracts from the surgeon's report are that on entering the abdomen the peritoneum was dusky colored, the cecum was dusky and somewhat cyanotic, as was the appendix and one inch of the ascending colon. The small bowel and the ileum were of normal color. There were no bands or adhesions. The surgeon states that he was unable to account for the color of the cecum and the appendix and also for the patient's high white count. There was no mention of any blood pressure fall following the operation, nor of any resuscitation being necessary. The pathological report revealed catarrhal appendicitis.

The mother's story, as elicited on October 9, 1943, was as stated, with the additional fact that on returning from the hospital the child was dizzy to the point of falling if someone did not support her. This symptom slowly disappeared after a period of two weeks. She further stated that the child's teachers, relatives and playmates

knew that her hearing was good prior to operation. The child recalled that her mother came to see her the evening of the operation and that she could not hear her mother speak. The child further stated that for some days following the operation she had pain in both ears, the type of which she was unable to describe, and that there was also noise in both ears similar to a steam radiator.

Findings of the physical examination were irrelevant except for those relative to the eighth nerves. Both membrana tympani were intact. They were normal in appearance and the canals were clean. Using the c^1 (256 d.v.) and the c^3 (1024 d.v.) tuning forks, the Weber test was referred to the left. The Rinne test was negative on the right side and positive on the left with both forks. Bone conduction was reduced twenty seconds with the c^1 (256 d.v.) fork and 28 seconds with the c^3 (1024 d.v.) fork on the left. The right side was reduced 14 seconds with the c^1 (256 d.v.) fork and 10 seconds for the c^3 (1024 d.v.) fork. There was no spontaneous or positional nystagmus. Caloric function tests with 30 cc. of water at 42° F. failed to induce a response in either ear. Twenty turns in ten seconds in the Barany chair likewise failed to induce a response in the canal of either ear. Fig. 1 shows the audiogram taken on November 6, 1943. Fig. 2 shows the audiogram of January 27, 1944. The masking apparatus was used in both tests.

A consultation report from the Neurosurgical Section revealed no abnormality except for the eighth nerve involvement. A patch test with avertin by the Allergy Section was negative. X-ray films of the skull, petrous bones and sinuses were normal. The blood Wassermann was negative, as was the urinalysis. It is apparent from the audiograms that the auditory acuity has remained almost stationary with the exception of increased bone perception in the left ear.

SUMMARY

A case of bilateral eighth nerve paralysis is presented in a twelve-year-old girl without other neurological symptoms following an appendectomy in which avertin-nitrous oxide-oxygen-ether anesthesia was used. The lesion appears organic in nature and the outlook for further recovery seems doutful. Neurological accidents following this type of anesthesia are not uncommon. Paralysis of both eighth cranial nerves without further brain damage, however, is extremely rare.

LOVELL GENERAL HOSPITAL.

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XXXV

FIBROMA OF THE ETHMOID AND FRONTAL REGION WITH CASE REPORT

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Memphis, Tenn.

Duncan G. Graham, M.D.
Sioux City, Iowa

Sam H. Sanders, M.D.
Memphis, Tenn.

Tumors in the region of the ethmoid and frontal sinuses are sufficiently common to be well known by all who do sinus surgery. However, it is believed that this case is worthy of being reported in detail, because of the tremendous size of the tumor, the technical difficulties attending its removal, including the problem of the management of exposed brain with or without dural covering, the surgical principle of complete exenteration of infected sinal mucosa that is emphasized, and the studies made during the postoperative period of the function of deglutition and phonation. By reference to the latter is meant motion pictures that were made of the superior and posterior aspects of the uvula, pillars, and pharyngeal musculature during the various stages of swallowing and of phonation in cooperation with the Department of Anatomy of the University of Tennessee Medical School.

REPORT OF A CASE

C. B., a colored male 63 years of age, was admitted to the John Gaston Hospital on January 17, 1943, with a history of having a growth in the upper part of his nose for five or more years. During the last two or three days there had been severe aching and pain in the left frontal region, with increased lacrimation in the left eye. Headaches had been so severe that he had to stop his work. He had not been able to breathe through the left nostril for a long time.

From the Department of Otolaryngology, University of Tennessee School of Medicine.

Adapted from a paper presented before the American Laryngological Association, New York, N. Y., June 8, 1944.

The patient stated that the left eye had been gradually pushed to the side. He had intermittent epistaxis during the last few months. He claimed to have had a similar tumor removed from his nose in 1933. However, reference to his previous record indicated only that nasal polyps were removed in 1933. He had no other operations nor any serious illness except the trouble with his nose. His family history was essentially negative for malignancy, tuberculosis, diabetes, and insanity.

Physical Examination. The temperature was 99° F.; pulse, 100; respiration, 20; and blood pressure, 120/70. The patient was a well-developed, rather poorly nourished elderly colored male who was not acutely ill but whose face was greatly deformed by the presence of a bulging mass situated between the eyes, extending upwards on to the forehead. The left eye was situated laterally and downwards from its normal position. The mass was firm, immobile, not tender, not nodular, and not attached to the underlying skin but fixed to the deep structures. The left eye was congested and hyperemic. He was able to count fingers with the left eye but its axis was markedly divergent from that of the right eye.

The bridge of the nose was widened and prominent and the nasal bones could be palpated. Anterior rhinoscopy revealed a mass that completely occluded the left side of the nose. The septum was deviated to the right, producing a partial obstruction of the right side. The mass seen in the left fossa was firm, red, smooth, slightly tender and slightly movable with an applicator.

The mouth and throat presented nothing of interest other than dental caries and poor oral hygiene. The ears were clinically negative with normal hearing bilaterally.

The lung fields were resonant and vascular. The heart was enlarged downward into the region of the P.M.I. in the sixth and seventh interspaces to the left of the midclavicular line. However, the heart sounds were clear and regular and there were no murmurs present.

The remainder of the physical examination was within normal limits.

The admission blood picture showed a mild secondary anemia. The urinalysis was negative. The Kahn test was negative.

X-ray films made the day after admission disclosed a destructive process involving the maxilla, malar and frontal bones on the left, as well as the ethmoids and the anterior part of the sphenoids.

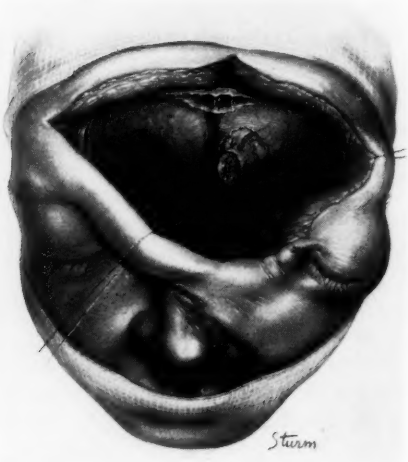

Fig. 1.—Drawing of post-operative field as viewed from in front.

On January 19, 1943, the mass in the left fossa of the nose was biopsied and the pathological report was probable fibrosarcoma with low-grade malignancy. Due to the magnitude of the tumor, it was not considered to be operable.

However, at this time the patient developed fever and pain, with increase in the size of the bulging mass. This became fluctuant very shortly and believing that an abscess was present an incision was made and drainage was carried out. The incision through the skin and subcutaneous tissues was made over the most prominent part of the fluctuation, situated midway between the center of the nose and the inner canthus of the left eye, and a large amount of thick yellow pus was evacuated. After the pus was evacuated, the anterior surface of the tumor was observed to be smooth, glistening, apparentlly encapsulated, and we considered at this time the possibility of enucleation of the mass.

The patient's general condition improved following drainage of the abscess but culture of the pus was positive for staphylococcus aureus and small gram-negative bacilli, morphologically Koch-Weeks bacilli.

On January 30, 1943, the tumor mass was removed in toto. It was attached to the dura by a pedicle from its posterior superior

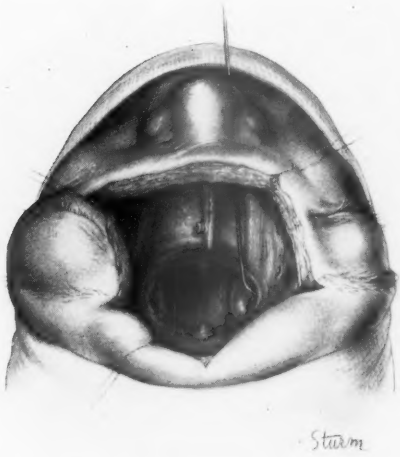

Fig. 2.—Drawing of post-operative field as viewed from head of table.

aspect, in the region of the posterior ethmoids. The dura was removed with the tumor over an area of about two inches in diameter overlying the left frontal lobe. There was a true retrobulbar abscess which was evacuated and drained. All of the sinuses on both sides of the nose were in a suppurative condition. Sulfanilamide was dusted into the wound after hemostasis was secured and the cavity was packed with iodoform gauze.

Pathological Report. Grossly the tumor was yellowish-white in color with grooves and ridges on its surface, corresponding to the sulci and gyri on the surface of the brain with which it had been in contact. It was well encapsulated and attached by a pedicle, apparently to the dura. On the cut surface it had a yellowish-white, whorled appearance almost identical with that of a uterine fibroid. The cut surface tended to bulge forward. Numerous small calcified areas were visible on this surface. The consistency was very tough.

Microscopic section showed numerous wide, swirling bundles of fibrous connective tissue. Isolated areas were more cellular, however, and in these were spindle-shaped cells. These more cellular areas were scattered at random throughout the section and had little regularity. Some of the sections were covered by a pseudostratified

columnar ciliated epithelium while others had a simple mesothelial surface.

The postoperative course was uneventful, except that the area of the exposed brain where the dura had been removed gradually bulged forward and downward into the cavity. Within a short time healthy granulations covered all the raw areas. The cavity reduced itself to approximately half its size, and the contents of the left orbit gradually swung back towards the midline. Vision of the left eye gradually improved until he could see almost as well with the left eye as with the right.

Considerable pus exuded from the remnants of the frontal sinuses, the sphenoid and maxillary sinuses. On March 30, 1943, the remnants of both frontal sinuses and the left antrum were exenterated.

On May 1, 1943, the sphenoidal sinuses were exenterated. During the course of this procedure the internal carotid artery on the left was injured. Profuse hemorrhage resulted, but this was quickly and easily controlled by means of an iodoform pack introduced into the sphenoid sinuses and filling the cavity. On May 6, the packing was removed and hemorrhage again resulted so it was repacked.

On May 9, 1943, hemorrhage resulted spontaneously while the patient was straining at stool. He was immediately taken to the operating room, the sphenoid repacked, and the left internal carotid artery ligated just above the bifurcation of the common carotid. The postoperative course has been uneventful since that time, and there has been no further hemorrhage. The packing in the sphenoid was left in situ for three weeks; the loose packing of the large cavity formerly occupied by the tumor was changed at frequent intervals. No bleeding from the sphenoid resulted when the packing was loosened and gradually removed three weeks after ligation of the left carotid artery (approximately six weeks after the original injury to the artery).

899 Madison Avenue

4014 Perry Way

1089 Madison Avenue.

Society Proceedings

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Meeting of Monday, January 3, 1944

THE PRESIDENT, DR. SAMUEL J. PEARLMAN, IN THE CHAIR

Symposium on Ménière's Symptom Complex

Otolaryngology

JOHN R. LINDSAY, M.D.

The term Ménière's syndrome has been used in connection with a group of diseases with paroxysmal vertigo, tinnitus and deafness which could not be explained on the basis of inflammatory disease, tumor or trauma. Clinical and pathologic observations have more recently made it possible to distinguish several entities within this general group. The term "Ménière's disease" has recently been used to denote a clear-cut clinical syndrome of recurrent attacks of vertigo, tinnitus and deafness. The inner ear pathology in this group is fairly well established as labyrinthrine hydrops. The term "pseudo Ménière's" has been used to denote vertigo attacks without auditory symptoms and without other neurologic signs. This clinical group is comparatively common and serves to complicate the diagnostic problem. The problem of the otologist in any case of dizziness is:

1. To determine whether the vertigo is of the vestibular type.

2. To determine whether it is located in the peripheral vestibular apparatus (including the eighth nerve) or whether it is central.

3. To make a diagnosis.

4. To elicit evidence which may help localize a lesion of the central nervous system.

Dizziness of nonvestibular origin can usually be differentiated on a careful history. The complete syndrome of tinnitus, deafness and vertigo indicate a peripheral lesion. In case of vertigo without auditory symptoms it is difficult to localize the site of the disturbance. Postural vertigo is extremely common in this group. Special examination for postural nystagmus may give information that will help differentiate between peripheral and central disease. Postural nystagmus, according to Nylen, is of two general types: *Type I*: where the nystagumus changes direction in different positions or while the patient is in the same position; *Type II*: where the direction is constant.

Clinical observations indicate that Type I occurs only in central nervous system lesions. Type II may occur in either peripheral or central lesions. Positional nystagmus in purely peripheral lesions is not common. Three cases were described: 1.) early Ménière's disease; 2.) toxic neuritis or labyrinthitis; 3.) vertigo in connection with catarrhal otitis media. All had positional nystagmus Type II. Four other cases of postural vertigo were described which fall into the groups sometimes classified as pseudo-Ménière's disease—one in a case of hypertension and arteriosclerosis, and three in "hypotensive stage" characterized by very low and variable blood pressure. In the latter four cases the postural nystagmus was always Type I, indicating that the vertigo was probably of central origin and that a true Ménière's disease could be ruled out. Such attacks are usually infrequent and sometimes not repeated.

In cases of positional nystagmus a labyrinthrine fistula must always be ruled out.

Conditions which have been commonly classed as Ménière's syndrome include vertigo due to 1) catarrhal middle ear changes, which is rare; 2) allergy, also infrequent; 3) hemorrhage and thrombosis, distinguished by the extensive permanent damage to function, the gradual recovery from the vertigo and the absence of recurrence; 4) toxic neuritis or labyrinthitis, also diagnosed on the characteristics of the attack; 5) Ménière's disease (labyrinthine hydrops). This may start with auditory symptoms such as tinnitus or deafness or as a simple attack of vertigo, but cannot be diagnosed until the complete syndrome is present. The auditory threshold varies widely in different stages of an attack. Bone conduction is depressed along with air conduction. Postmorten examination has demonstrated a labyrinthine hydrops as the basic pathologic entity. The etiology is still indefinite.

Medical and neurosurgical treatment were not discussed. A small percentage of cases require surgical intervention to prevent the vertigo. In these a surgical procedure that will be universally satisfactory must preserve the hearing. Up to the present the only procedures on the labyrinth that have met these requirements are Portmann's operation and a modification of the fenestration operation. Neither has as yet been universally satisfactory.

Neurosurgery

ERIC OLDBERG, M.D.

In 1861 Ménière first described the syndrome, which bears his name, of tinnitus, deafness, and paroxysmal attacks of vertigo. In 1874, Charcot hypothesized that since these patients tended to recover when they became totally deaf, division of the acoustic nerve might be of therapeutic benefit. This was again suggested in 1894 by Sir Charles Ballance. The first recorded division of the eighth nerve was done in 1905 by Krause, who, however, performed the operation for tinnitus rather than for Ménière's syndrome. In 1908, both Ballance in England and Frazier in the United States cut the eighth nerve for Ménière's disease. In 1928, Dandy suggested that the eighth nerve could be sub-totally sectioned, sparing the auditory fibers and dividing only the vestibular fibers. The first man to put this suggestion into practice was McKenzie in Toronto in 1931, and in 1933 Dandy began differential sections —now the accepted surgical procedure for the condition. In 1932, an alternative operation was proposed by Putnam, by which the middle fossa and petrous ridge were exposed, and the vestibular ganglion electrically coagulated. The latter operation, however, has not been greatly utilized.

The syndrome is characteristically present in middle and later life and is more common in males than in females, and occurs more often on the left than on the right side. Before entertaining ideas of surgical intervention, a differential diagnosis should be made from labyrinthitis or cerebellopontine angle tumor. If, in spite of adequate medical therapy, including a salt-free diet, the use of histamine, and nicotinic acid, the attacks are so violent and so frequent over such a period of time that the patient is seriously disabled for gainful occupation, then the idea of surgical intervention must be entertained, unless total deafness is rapidly intervening and offers some hope of spontaneous cure. Differential section of a half or

slightly more than a half of the eighth nerve is the operation of choice, and this can usually be done so as to section a minimum of cochlear fibers, and a maximum of vestibular fibers. Whereas the operation should carry a very low mortality risk, it nevertheless is a major procedure, since the cerebellopontine angle must be exposed. In view of the comparative frequency of vascular anomaly in this area, the morbidity risk with special reference to the facial nerve is higher than the mortality risk. Operation may usually be expected to stop the attacks of paroxysmal vertigo. However, it does not, of course, stop the progress of deafness, and in a fairly large percentage of cases it does not stop the tinnitus. There are usually no disabling postoperative complications, such as diplopia or positional vertigo, in the average case, although these sometimes occur, especially where a bilateral operation has been performed.

In conclusion, and in order to indicate the wide variety in philosophy of treatment, it may be stated that at approximately the same date (1940-41) 20 such operations had been performed in one large clinic, and 401 in another clinic of considerably less size.

Medicine

BAYARD P. HORTON, M.D.

ROCHESTER, MINN.

Ménière's symptom complex is characterized by recurrent attacks of sudden, severe vertigo, nausea, vomiting, tinnitus and deafness of unknown etiology. It constitutes a definite clinical syndrome. Numerous forms of therapy have been suggested since the syndrome was first described by Ménière in 1861; however, the perfect treatment has not yet been announced.

My interest in the problem began with the introduction of histamine therapy. Shelden and I,[1] in January, 1940, stated in our first communication regarding histamine therapy that "our purpose in presenting this form of therapy is to make available to the medical profession a quick and ready method for controlling the acute symptoms of Ménière's disease." At that time we had in mind, primarily, the vertigo of Ménière's disease. Since this original publication a large number of patients having Ménière's symptom complex have been treated at the Mayo Clinic.[2] I still consider that intravenous histamine therapy is the best method for controlling the

acute symptoms of this disease. (A detailed report of the investigation of these cases will be published later after this further experience.) I realize, however, that it is extremely difficult to evaluate any type of treatment for Ménière's syndrome because many patients make spontaneous recoveries from this disease entity.

Vertigo is the essential feature in Ménière's symptom complex and represents a local process involving the labyrinth. The presence of tinnitus and varying degrees of deafness probably indicate that the process also involves the cochlea. The process most likely responsible for Ménière's syndrome is local alteration in the permeability of the capillary wall with secondary local edema.

During acute attacks the majority of patients have objective vertigo, and it is interesting to note that a higher percentage of patients who have this type of vertigo derive benefit from treatment with histamine than do those patients who have subjective vertigo. Why this is true is not clear at present.

Although vertigo is the essential feature in Ménière's disease, tinnitus is also an annoying complaint and a difficult one to treat. Tinnitus appeared simultaneously with the onset of vertigo in 45 per cent of the cases of Ménière's disease studied. It preceded the vertigo in approximately 28 per cent and followed vertigo in 21 per cent. In the remaining 6 per cent, the exact time of onset of tinnitus could not be determined. The onset of some degree of deafness occurred simultaneously with the onset of vertigo in 48 per cent of cases. It preceded vertigo in 38 per cent and followed vertigo in approximately 14 per cent.

If patients are treated promptly with histamine given intravenously during the initial acute attack, it seems to be possible to prevent deafness in a large percentage of cases. Patients so treated in and around Rochester have had little or no loss of hearing.

Dr. B. E. Hempstead and I have recently observed a patient aged 46. Complete loss of hearing in her right ear began with the initial attack of vertigo. Intravenous histamine therapy was started approximately 72 hours after onset of the attack. Following 16 daily intravenous injections of histamine (12 were given in 0.8 per cent solution of potassium chloride and the remaining 4 in 0.9 per cent solution of sodium chloride), she obtained not only complete relief of vertigo but the hearing in the right ear improved to within almost normal limits. The hearing in the left ear was entirely normal and remained so.

In my opinion, the most important point to keep in mind when confronted with a patient who has acute symptoms is the need for prompt institution of treatment in the attempt to prevent loss of hearing. As stated before, I believe loss of hearing can best be prevented and the acute attack aborted by intravenous administration of histamine.

The second patient was a physician's wife, aged 38. When she was observed first, she complained of nausea, vomiting, tinnitus and marked loss of hearing in the left ear. She received histamine intravenously by the usual method, on November 23, 24 and 27, 1939. At the completion of the first intravenous injection of histamine she was able to hear moderate tones in the left ear and the tinnitus had lessened approximately 75 per cent. Since she was the second patient treated in this manner, she was allowed to go home and we awaited future developments. She was seen again February 23, 1940, with an acute attack of vertigo, tinnitus in the left ear, nausea, vomiting and a sensation of pressure in the head. Intravenous histamine therapy was started immediately, the nausea disappeared in approximately eight minutes, the bed stopped "whirling" within fifteen minutes and the tinnitus disappeared at the end of an hour. She was then able to get up and walk without assistance. Her husband has continued to give her daily subcutaneous injections of histamine of approximately 0.05 to 0.067 mg. histamine base and she has remained free from attacks up to the present.

Many patients with Ménière's symptom complex have a horror of these attacks. The fear of vertigo seems to incapacitate as many patients as does vertigo itself. Many physicians do not seem to appreciate this fact.

It has not been possible to distinguish between the so-called "histamine sensitive" and the "histamine insensitive" types of Ménière's symptom complex as suggested by Atkinson of New York. The problem is not that simple. Anyone interested in the subject of histamine skin tests will do well to read Browne's[3] article which is an abridgement of a thesis for which the work was done in my laboratory.

Misconceptions have arisen in the minds of the medical profession regarding intravenous histamine therapy. The method of giving 2.75 mg. histamine diphosphate intravenously in a 1:250,000 dilution of normal saline is a simple procedure, and at the Clinic a large number of such injections have been given to patients with Ménière's symptom complex without any untoward effects. Fur-

thermore, the majority of these patients have been ambulatory, as hospitalization was not necessary.

One of my associates, Dr. Peters, suggested several months ago that histamine diphosphate (2.75 mg. histamine diphosphate) be given in 0.8 per cent solution of potassium chloride instead of 0.9 per cent solution sodium chloride. This procedure has been carried out in many instances. Whether there is any advantage in isotonic solution of potassium chloride over isotonic solution of sodium chloride has not been determined as yet.

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2. Horton B. T.: The Use of Histamine in Ménière's Disease, Surg. Gynec. & Obst. 72:417-420 (Feb. 15) 1941.

3. Browne, H. C.: Cutaneous Reactions as an Index to Desensitization to Histamine, J. Allergy, 14:19-48 (Nov.) 1942.

DISCUSSION

DR. ALFRED LEWY: The terms Ménière's syndrome, Ménière's disease, pseudo-Ménière's disease are, I believe, very confusing. We have no clinical correlation, as proved by remedial measures, to indicate that we know what we are dealing with in many cases. There is no doubt that many cases are due to ectasia of the membranous labyrinth, but until the diagnosis is proved by remedial measures in a considerable number of cases, I do not think we should give it a definite name. I believe we should observe the patient from the standpoint of the vertigo, and then rule out one by one the various things that may produce vertigo. I also object to the term "toxic labyrinthitis." Some of the cases I have seen that were diagnosed as toxic labyrinthitis were the result of encephalitis.

Mention has been made also of the Bárány test. I have recently observed a patient, a young man about to be inducted into the army, who had been seen by a neurosurgeon who believed he had a functional disturbance. When I saw him I found definite postural nystagmus and a perverted caloric nystagmus. I returned him to the neurosurgeon and said I believed he had an organic lesion in the posterior fossa. He was again hospitalized, although the neurosurgeon thought it was a psychogenic affair, but eventually found evidence of a cerebellar tumor which was removed. If we appreciate the condition from the standpoint of vertigo, I think we are less

apt to go astray in the matter of treatment for this so-called Ménière's syndrome.

Dr. Leonard Cardon: Injections of histamine phosphate are used to produce experimental peptic ulcer in animals. I would like to know if there is any clinical evidence that this danger is present in patients being treated for Ménière's or other syndromes by repeated injections of histamine phosphate.

Dr. John R. Lindsay (Closing): I enjoyed Dr. Oldberg's discussion of the neurosurgical aspect of this problem, and also Dr. Horton's remarks, especially his statement to the effect that they had only proposed histamine treatment to relieve or abort an acute attack. In our experience with histamine this has been the only desirable result obtained. Unfortunately we very seldom see a patient in the acute stage of an attack, and therefore have not often been able to evaluate it in that respect, but we have not been able to prevent attacks.

One reason why I pointed out the wide fluctuations in the hearing, was to indicate why one cannot evaluate therapeutic effects on the basis of an alteration in the hearing threshold. I have never seen a patient with a 40 or 50 decibel loss return to normal. If a patient has lost his hearing completely and then recovers it entirely, there is a question of the diagnosis of Ménière's disease.

With regard to Dr. Lewy's statement about toxic neuritis versus toxic labyrinthitis, it can only be said that we do not know the exact pathologic process or its limitations. One reason why I showed cases of postural vertigo without auditory findings was because it is my impression from the literature that many cases are improperly classed as Ménière's disease when the vertigo may be of central origin. Some of these cases never have a recurrence; others may have a few but it is difficult to see any justification for operation. I think it is of considerable help in diagnosis of this particular group of diseases to make a routine investigation as to whether they have postural nystagmus.

Dr. B. T. Horton (Closing): We feel that the intravenous administration of histamine (2.75 mg. histamine diphosphate in 250 cc. physiologic saline solution, administered at the rate of 30 to 90 minims per minute, depending upon the tolerance of the patient) is a safe and efficient form of therapy. It will abort the acute symptoms of this disease in the majority of instances. It is not necessary, except in rare instances, to hospitalize patients for this procedure.

Since we began this type of experimental therapy in 1939 we have given approximately 14,700 such injections to patients of various types, including those with Ménière's symptom complex and multiple sclerosis, without any ill effects except in one case, that of a man aged 20. He had multiple sclerosis and he developed an acute gastric ulcer after 13 injections had been administered. A roentgenogram of the stomach showed an ulcer on the lesser curvature, which healed completely within 12 days after intravenous histamine therapy had been discontinued.

In our routine procedure where patients are receiving daily intravenous injections, such as occurs in our experimental work with reference to multiple sclerosis, it is important to have them eat a full meal immediately before administration of the drug. This particular patient had failed to do so. When histamine is administered it produces a maximal rise in gastric acidity; hence it is important to have food in the stomach to absorb the excess gastric acids. It is interesting to note that during our experimental work we have given more than 328 such injections in a given individual having multiple sclerosis without producing any measurable ill effects.

Dr. Lindsay has discussed the pathology of Ménière's symptom complex, and I do not feel that I can add anything to his discussion. As I have previously indicated, the factor most likely responsible for Ménière's symptom complex is local alteration in the permeability of the capillary wall with secondary local edema of the labyrinth.

Meeting of Monday, February 7, 1944

THE PRESIDENT, DR. SAMUEL J. PEARLMAN, IN THE CHAIR

Laryngeal Management of Bulbar Poliomyelitis

THOMAS C. GALLOWAY, M.D.

(Abstract)

In respiratory difficulty of acute poliomyelitis, emphasis has been placed on central factors. Little attention has been paid to what happens in the peripheral respiratory tract.

In cases of bulbar poliomyelitis accumulated salivary and other secretions amounting to 1500 to 2000 cc. per day may lie as a pool over or in the airway, as seen by direct laryngoscopy, and may impede or prevent inflow of air with production of serious anoxia; or it may be drawn forcibly into the lung bed, especially if a respirator is used, producing asphyxia, obstruction and such sequelae as atelectasis or pneumonitis. Or this secretion may produce serious derangements of coordination and rhythm of respiration, some of which are probably reflex since they occur in unconscious patients and are relieved when the secretion is removed.

Anoxia is probably important and seems to account for restlessness, depression and other symptoms, sometimes ascribed to the infection itself, since these are strikingly relieved when the anoxia is corrected.

Most patients with bulbar poliomyelitis will recover if treated early by oxygen, postural drainage, aspiration and parenteral fluids, and the respirator if needed. Two cases are cited, however, in which the patients were saved only by tracheotomies which prevented immediate asphyxiation, short-circuited secretion and allowed its aspiration, and relieved serious anoxia. The respirator was very satisfactorily employed in one of these after tracheotomy. This measure may well be more widely used before a critical stage is reached and should considerably lower the mortality from this disease.[1]

REFERENCE

1. Galloway, T. C.: Tracheotomy in Bulbar Poliomyelitis, J. A. M. A. 123: 1097-98 (Dec. 25) 1943.

DISCUSSION

DR. ALFRED LEWY: I think we have listened to an historic presentation. I do not know and am not familiar with the literature on the subject, and I do not know whether or not Dr. Galloway was the first to present this suggestion, but this is the first I have heard of it. The idea that the chief complication in bulbar poliomyelitis is due to anoxia is very important. We often lose sight of the fact that all deaths are due to lack of oxygen by circulatory or respiratory failure, and our first effort should be to get oxygen to that part of the system that needs it. Vital centers do not live long without oxygen.

Some years ago I advanced a method of draining the lung in lung abscess by passing a catheter through the Mosher life saver

which has had an opening made in the bottom. This can be passed down into the right or left bronchus. It seems to me that this technic might be used pending tracheotomy, in such cases as Dr. Galloway described.

DR. FREDERICK HILLER: I am sure we have all been impressed by this contribution to the treatment of infantile paralysis. I myself have seen the life-saving effect of tracheotomy in one of our patients. There is no doubt that tracheotomy is indicated in certain cases of respiratory paralysis, those with obvious obstruction of the air passages and disturbance of the physiologic separation of breathing and swallowing. Both indications may become urgent for patients in a respirator, more so than without it.

Obstruction of air passages may occur in spinal as well as in bulbar paralysis, in the former because of motor inability to expectorate, in the latter because of disturbance of the cough reflex. The effect of passive respiration under negative pressure can be very unfortunate in both types of obstruction. Harper and Tennant noted ten years ago that "the forced sucking in of air often caused aspiration of material from the throat and at the same time overcame the reflex coughing and choking by which such material is usually ejected." Even if one restricts the use of the respirator entirely to treatment of spinal respiratory paralysis, the danger of aspiration followed by pneumonia remains. In Landon's well-known statistics based on 2,402 cases of poliomyelitis in 1931, 16 of 35 respirator treated patients died within 18 months from respiratory infections, chiefly pneumonia. Should a vagus glossopharyngeal paresis be added to the difficulties of respiration the danger of aspiration becomes much greater. "The accumulation of secretion and food around the glottis may produce a rapid shallow type of breathing closely simulating intercostal paralysis." (J. L. Wilson). Vagus paralysis and the action of the respirator act together in abolishing the cough reflex and helping the accumulation of large amounts of fluid in the pharynx and the larynx, which interferes gravely with respiration.

All who have had personal experience with treatment of respiratory failure will agree that the differentiation of spinal from bulbar types is not always as simple as the textbooks would have us believe. Ascending types of paralysis may appear to be of spinal type in the beginning, only to become more bulbar after a day or so. No one would take such patients out of the respirator; one feels that they must be kept breathing to keep them alive. But breath-

ing with aspiration from the pharynx means serious pulmonary complications, and insufficient respiration through obstructed air passages means steadily increasing anoxemia. Anoxia of the tissues shows itself in impaired function of the brain as a whole, particularly in drowsiness progressing to coma; there is also a detrimental effect upon the already depressed action of the respiratory center. Autopsy findings showing organic lesions in the gray, and especially in the white, matter of the central nervous system should always be regarded as possible effects of anoxia when the well-known symptoms of true inflammation are absent. There seems to be little doubt that many of the so-called encephalitic symptoms, as well as manifestations of lesions of the pyramidal tract, are the cause of tissue anoxia and not of localization of the poliomyelitis process.

Oxygen supply to the tissues is required in such cases, and tracheotomy should be considered. If there is a chance that oxygenation of the blood may save the patient's life, tracheotomy may do so. The patient will regain consciousness and the damaging effects of anoxemia on the respiratory center will be overcome. Tracheotomy will fail in the severe cases of bulbar poliomyelitis in which the patients die from combined respiratory and circulatory failure. We know that in cases of drowning or electric shock the heart will continue to beat for several minutes after respiration has ceased. In bulbar poliomyelitis, a heart that has been beating strongly, regularly and slowly under the effect of oxygen stimulation will fail rapidly under the combined effect of anoxemia and the inflammatory lesion of the circulatory center. This has been shown in animal experimentation; when the carotid sinus and the aortic nerves have been severed, thereby eliminating a great part of the nervous control of the circulation, the animal will die with a loss of only one-tenth of its blood volume. The abolition of function of the medullary, circulatory and respiratory centers is, of course, a much more severe shock to the maintenance of circulation than the cutting of the buffer nerves, and the amount of blood withdrawn from the circulation by the peripheral loss of vascular tone exceeds by far the ten per cent loss which will kill the experimental animal.

I do not think that bulbar respiratory disturbance per se is a contraindication to artificial respiration, provided the air passages have been kept free. At least one of Dr. Galloway's cases proves this point. Oxygen may help over a critical situation.

Dr. Thomas C. Galloway (closing): Dr. Hiller was of great assistance in these cases. I wish we had time for him to discuss the histologic picture in poliomyelitis, and especially to discuss

the damage to brain and heart tissue from anoxia as contrasted with that from the virus infection.

Dr. Lewy's remarks were very kind. I think his idea of the Mosher life saver for aspiration might be very valuable in these cases.

The So-Called Crypt System of the Human Pharyngeal Tonsil—A Study of its Development, Adult Structure, and Relations

L. B. Arey

Department of Anatomy, Northwestern University Medical School

(Abstract)

A difference of opinion exists concerning the morphology of the pharyngeal tonsil. Many writers hold that the palatine, lingual and pharyngeal tonsils are all essentially alike in structure. Others stress the peculiar mucosal folding in the pharyngeal tonsil but deny crypts to it. However, up to the present time the exact morphology of the pharyngeal tonsil has not been determined through the method of reconstruction, as have the palatine tonsil (Minear, Arey and Milton) and the lingual tonsil (Hellman).

A pharyngeal tonsil of a newborn has been studied and reconstructed. A distinctive feature is the folding of the mucosa into roughly parallel ridges and troughs. Also prominent are the numerous pores that open from the free surface of the ridges and troughs into relatively large and long epithelial funnels. The latter are embedded in lymphoid tissue. The unbranching funnels resemble somewhat the simplest crypts of the palatine tonsil, but, unlike the palatine tonsillar crypts, each usually communicates with subtonsillar glands. Closer is the resemblance to the simple pits of the lingual glands, which do have glands opening from their depths. It might be argued whether the funnels of the pharyngeal tonsil are the dilated ducts of glands or whether they are simple crypts that have budded off glands. In either case the morphologic appearance and lymphoid relationship is crypt-like in the accepted tonsillar sense.

Dr. Walter Theobald: I rise to compliment Dr. Arey on the tremendous study this brilliant presentation represents. The

tonsil subject is several hundred years old, but one can still enjoy new studies such as those so well shown here. I should like to have some clarification on one matter. Dr. Arey did not say much about the capsule, and rightly so, because he was talking about crypts. I remember that Dr. Eikelson asked—has the tonsil a capsule or is it a fascia? In the operating room the experts talk about the capsule when removing tonsils. I wonder if he can tell us whether he considers it a capsule or an aponeurosis.

DR. L. B. AREY (closing): I think the answer to Dr. Theobald's question depends on definition. What is a capsule? I see no reason why a capsule cannot be a fascia. What is the capsule of the thyroid? It is part of the cervical fascia; it is a region where a gland has developed, has enlarged, has pressed on and compressed the tissue around it. As a result, the compressed tissue has been forced to consolidate into a capsule. If you look at the stages of development of a palatine tonsil, you will be again impressed by the fact that as the tissue gets bigger it presses on the underlying fascia; the result is that the so-called capsule of the tonsil is nothing but a fascial region that has been subject to pressure. I do not see any reason why one should question the propriety of a capsule developing in that way. From such a capsule in the palatine tonsil you have connective tissue which extends inward between the folds of lymphoid tissue and flattens into the septa or trabeculae of the tonsil. I do not look upon it as unorthodox to have capsules in trabeculae develop in that way or feel that one should apologize in speaking of such an origin for these structures.

There is one point I failed to mention in my talk. Many workers have reported the presence of cystic, epithelial structures in the pharyngeal tonsil. They have been reported as being pathologic things, which they are, but related to ducts. What is the genesis of such cystic cavities? The point which I think has been missed is that the whole process of dilatation is already there from early embryologic life. The actual facts and findings of cysts are well known, but the thing that has not been known is that the forerunner of the cyst is the dilated duct which is a morphologic structure, perfectly normal from the early months onward.

Abstracts of Current Articles

NOSE

Sulfadiazine in the Treatment of the Common Cold.

Cecil, R. L., Plummer, N., Smillie, W. G.: J. A. M. A. 124:8 (Jan. 1) 1944.

A controlled study of the effect of Sulfadiazine given orally on the common cold is presented.

Serial blood studies, nasopharyngeal cultures, oral temperatures, and nasopharyngeal examinations were obtained in both the 48-patient experimental group and 24-patient control group.

Sudfadiazine was administered orally in a dosage of one gram three times daily for four days, resulting in blood concentrations between 3.4 and 10.5 mg. per hundred cc.

In normal individuals a prompt and marked reduction in normal nasopharyngeal flora was observed when the blood level of sulfadiazine reached 4-6 mg. per hundred cc., but the flora rapidly returned to its former prevalence and distribution two to three days after the drug was discontinued.

In the untreated control group the bacterial content of the nasopharynx was observed to drop temporarily during the "watery" stage of a cold and thereafter to increase rapidly.

When sulfadiazine was given during the course of a cold, however, cultures showed a uniform decrease in the number of organisms and a check in the growth of pathogens.

The clinical course of the treated cases showed no striking differences from that of the controls; however, there appeared to be some amelioration of symptoms due to control of the secondary infection.

The routine use of sulfonamides in the treatment of the common cold is discouraged except in a few selected cases as a protection against severe secondary infection.

Sooy.

LARYNX

Chronic Laryngeal Stenosis.

Moore, P. M.: Cleveland Clinic Quart. 11:5 (Jan.) 1944.

Successful operative treatment of a subglottic stricture in a 27-year-old male by means of a split thickness graft and foam rubber mold is described.

Figi's modification of Arbuckle's method was used.

The stricture was preceded by multiple removal of laryngeal papillomata and by a previous tracheotomy.

Previous bronchoscopic dilatation and electro-resection were unsuccessful.

SOOY.

Post-Thyroidectomy Laryngeal Paralysis (Bilateral).

Holinger, P. H.: South. M. J. 37:169 (Mar.) 1944.

Holinger quotes Work in stating that in 212 cases of laryngeal paralysis, 84 followed thyroidectomy and 14 were bilateral. The narrowing of the glottic chink is aggravated by myxedematous infiltration of the vocal cords. Parathyroid deficiencies are likewise noted in these types of patients. The apparent cause of the triad of symptoms is the extensiveness of the thyroidectomy in which too large an amount of the gland is removed, including the superimposed parathyroid glands, accompanied by a severance of both recurrent laryngeal nerves. He mentions the greater reliability of the symptoms and physical signs of hypothyroidism as compared with the inaccuracy of the basal metabolic rate because of the interference with respiration, even with a tracheotomy tube in place.

The correction of these deficiencies lies in the administration of the glandular substances in which the patient is deficient. The surgical measures mentioned for establishing greater airway are tracheotomy, the removal of a vocal cord by various means, lateral transfixion of the arytenoids, and widening of the angle of the thyroid cartilages by the interposition of implanted cartilage between the alae. The importance of preserving the voice in any surgical procedure is stressed.

MCMAHON.

BRONCHI

Sulfonamides in Bronchial Secretion. The Effect of Sulfonamides in Bronchiectasis.

Norris, C. M.: J. A. M. A. 123:667 (Nov. 13) 1943.

A method of preparing a clear filtrate from bronchial secretions for sulfonamide concentration is described.

Ten patients with bronchiectasis were studied and all secretions were obtained bronchoscopically.

The sulfadiazine concentration in the bronchial secretions approximates 60 per cent of the blood concentration when the drug is administered orally. This fraction is not affected by the amount of secretion or by the extent of the disease.

When 5 per cent aqueous suspension of microcrystalline sulfathiazole was instilled into the trachea, significant concentrations were found to persist in the bronchial secretions for 24 to 48 hours. This concentration was materially diminished in the presence of large amounts of secretion.

Two and one-half per cent aqueous solution of sulfadiazine instilled intratracheally is almost entirely eliminated in 24 hours.

SOOY.

Roentgen Diagnosis of Bronchiogenic Carcinoma.

Shinall, H. L.: Radiology 42:213 (Mar.) 1944.

Forty autopsied cases of bronchiogenic carcinoma seen over a five-year period were studied. Roentgenograms showing a unilateral rounded nodular shadow, usually in the central zone of the lung, coupled with six points in the clinical history, gave a correct diagnosis in 87 per cent of the cases. These clinical points are: age over 40 years, duration of symptoms over 3 months, dry cough, expectoration of blood, loss of weight and lack of fever.

JORSTAD.

ESOPHAGUS

Carcinoma of the Esophagus in Association with Achalasia of the Cardia.

Bersack, S. R.: Radiology 42:220 (Mar.) 1944.

In only one of 227 cases with carcinoma of the esophagus admitted to Hines Hospital between 1931 and 1942 was achalasia associated. Literature reveals no statistical etiological correlation of carcinoma to achalasia. Importance of evacuating the contents of the esophagus prior to roentgen examination is emphasized.

JORSTAD.

Lesions of the Esophagus in Generalized Progressive Scleroderma.

Lindsay, J. R., Templeton, F. E., and Rothman, S.: J. A. M. A. 123:745 (Nov. 20) 1943.

The edematous, indurative, and atrophic stages of generalized progressive scleroderma are discussed.

Difficulty in swallowing was reported in 16 cases of diffuse scleroderma. Esophagoscopy in one instance revealed a dense fibrous stricture at 32 cm. without ulceration. Postmortem findings in another case showed esophageal dilatation to 4.5 to 5 cm. in width and absence of the mucosa over the lower three-fifths of the organ.

Five consecutive cases of clinically typical diffuse scleroderma with esophageal symptoms are presented.

The esophageal disturbances consisted of difficulty in swallowing solids or liquids, especially in the recumbent position, because of delayed emptying of the esophagus. Frequently also a burning pain was noticed behind the sternum which appeared about an hour after meals and was worse on lying down and particularly when lying on the left side. This is felt to be due to the regurgitation of gastric contents into the esophagus and a resulting chronic esophagitis.

Barium studies revealed a loss of peristalsis in the lower two-thirds of the esophagus with relaxation and mild dilatation in this area as low as the phrenic ampulla. Some atony of the cardiac sphincter was also felt to be present.

Esophagoscopy in four cases revealed ulceration in the lower third of the esophagus localized chiefly to the region just above the phrenic ampulla. Stricture formation in the region immediately above the phrenic ampula was observed in later stages.

These findings were attributed to the esophagitis with scleroderma-tic changes as a predisposing factor.

Biopsy in two cases revealed changes in the connective tissue of the submucosa suggestive of sclerodermatic change.

SOOY.

Dysfunction of the Hiatus Diafragmatico.

Grez, Anibal: Rev. de Otorinolaryng. 3:2-14 (June) 1943.

The author reviews his experience with the pathological condition commonly known as cardiospasm. No really descriptive name has been given to this disease, which in reality does not correspond to spasm, nor does it affect the cardia.

Normally peristaltic contractions of the esophagus carry the bolus to its lowest portion, which is opened by the coordinated action of the nerve plexus controlling deglutition and relaxation of the ring of muscle, which is part of the diaphragm surrounding the esophagus. Loss of coordination between the nerve plexus and the muscular ring results in so-called spasm or failure of the lower portion of the esophagus to open.

The diagnosis is made by the findings noted during the passage of the esophagoscope. The lumen is found to be closed and contracted but yields to gradual pressure, and the instrument is then easily passed into the stomach.

Other conditions which must be differentiated from functional contractions are such organic lesions as ulcers, esophagitis, tumors of the esophagus itself, or compression caused by inflammation or tumors of the adnexa. By means of esophagoscopy conditions which are difficult to interpret in the x-ray film have been correctly diagnosed.

If stenosis develops, regardless of the etiology there follows an hypertrophy of the muscle fibers immediately above it. Eventually this is followed by atrophy and dilatation which results in mega-esophagus. In this condition the picture, as seen in the esophagoscope, is described as follows: The lumen is large, the mucosa is pale, edematous, and in spots is covered by a thick fibrinous coat.

The symptoms vary according to the duration of the disease. In the early stages occasional regurgitation and vomiting are present. Later there is pain after swallowing and an increase in frequency of regurgitation and vomiting. Loss of weight then be-

comes apparent, and late in the disease retrosternal pain is almost constant. When ulcerations occur, the pain is very severe. Tracheobronchial compression may occur and is manifested by nocturnal cough.

The treatment is essentially dilatation. Various types of dilators have been used under visualization by x-ray. The authors have had brilliant results following Mosher's technique. After re-establishing function the treatments are repeated at intervals of about six months or longer as necessary.

HIGBEE.

MISCELLANEOUS

Vitamin B_1 Hypersensitivity With Desensitization.

Mitrani, M. M.: J. Allergy 15:150 (Mar.) 1944.

The author reports a case of allergy to vitamin B_1. The day following an injection of 50 mg. of thiamine hydrochloride there appeared on the face, chest and back of a patient a maculo-pruriginous eruption. A similar eruption recurred on reinjections of the thiamine. Sensitization was confirmed by intracutaneous and passive transfer tests. Desensitization was accomplished by the daily injection of 0.1 cc. of a thiamine hydrochloride solution with physiological saline solution in dilutions from 1:5000 to 1:10 until she could tolerate 1 cc. of a solution containing 100 mg. of thiamine for ten consecutive days, and the intradermal tests were negative.

MCMAHON.

The Prevention of Ear and Nasal Sinus Complications of the Common Cold.

Dolowitz, D. A., Loch, W. E., Haines, H. L., Ward, A. T., Jr., and Pickrell, K. L.: J. A. M. A. 123:534 (Oct. 30) 1943.

The authors feel that 2.5 per cent sulfadiazine in ethanolamines solution sprayed locally in the nose and throat is of value in reducing the incidence of complications from the common cold.

One hundred three nurses were studied and at the first sign of a cold were sprayed eight to twelve times daily for three days, then five to eight times daily for three days. Ethanolamine solution alone was used in the 44-patient control group.

Sinusitis as evaluated clinically was reduced from 30 to 9.7 per cent, otitis media from 4.5 to 1.8 per cent, sore throat from 10 to 0.0 per cent, laryngitis from 2.3 to 0.0 per cent, and cough from 44 to 0.0 per cent.

The sulfadiazine spray was effective in reducing the concentration of beta hemolytic streptococci cultured from the throat but had little effect on pneumococci or staphylococci.

SOOY.

Combined Penicillin and Heparin Therapy of Subacute Bacterial Endocarditis. Report of Seven Consecutive Successfully Treated Patients.

Loewe, L., Rosenblatt, P., Greene, H. J., and Russell, M.: J. A. M. A. 124:144 (Jan. 15) 1944.

Seven consecutive patients with subacute bacterial endocarditis were treated with the combined use of penicillin and heparin.

Six patients had a preceding rheumatic valvulitis and one, a congenital cardiac defect.

In five instances the organism was streptococcus viridans, the sixth was a streptococcus hemolyticus and the seventh a type 27 pneumococcus.

The penicillin was given by intravenous drip and the heparin was usually deposited subcutaneously.

The immediate effects were uniform sterilization of the blood and relief of clinical manifestations in all cases. In a few cases the efficacy of the treatment may have been enhanced by the preliminary use of sulfonamides.

Posttherapy management included removal of possible foci in the teeth and the nasopharynx. These proceedures were preceded and accompanied by additional penicillin therapy.

SOOY.

An Association Between Red-Green Color Blindness and Some Cases of Asthma and Hay Fever.

Molholm, H. B.: J. Allergy 15:120 (Mar.) 1944.

In order to arrive at a conclusion as to whether the higher incidence of asthma and hay fever in males (2:1) could be accounted for on the basis of a sex-linked factor, the author uses the criterion of red-green color blindness as a comparison. He expresses the

hypothesis that if the incidence of red-green color blindness in boys with asthma or hay fever were found to be significantly greater than among unselected males, then that finding would be taken as indirect, but adequate, evidence that some cases of asthma and hay fever depend in part on a sex-linked recessive factor.

He states that the incidence of red-green color blindness among unselected males is 4 per cent, according to the Ishihara tests. His results show that the incidence of red-green color blindness was highest among those patients whose first allergic symptom was asthma and was first experienced before the age of 13 years. Of those 104 patients, 13 were red-green color blind. This incidence of 12.5 per cent is about three times as great as that among unselected males.

From this foregoing statement, the author concludes that there is a relationship between red-green color blindness and some cases of asthma and hay fever which may indicate that those cases depend in part on a sex-linked recessive factor.

McMAHON.

Combined Heparin and Chemotherapy in Subacute Bacterial Endocarditis.

Katz, L. N., and Elek, S. R.: *J. A. M. A.* 124:149 (Jan. 15) 1944.

Four patients suffering from streptococcus viridans endocarditis were treated with heparin and chemotherapy consisting of either sulfonamides or sulfonamides and intensive arsenotherapy.

There was no evidence of clinical recovery and two of the four patients showed evidence of cerebral hemorrhage during the heparin therapy.

SOOY.

Histological Effects of Sulphonamide-Proflavine Mixtures in the Rabbit. Some Experimental Observations.

Russell, D. S., and Beck, D. J. K.: *Brit. M. J.*, Jan. 22, 1944, p. 112.

The effect of sulphonamide-proflavine mixtures on tissues is observed experimentally in rabbits and the following conclusions are reached:

1. Insertion of substantial amounts of sulfathiazole powder into a clean wound in the abdominal wall is harmless to tissues and is largely absorbed by the tenth day.

2. Similar amounts of powder consisting of 1 part proflavine sulphate to 100 parts of sulfathiazole caused considerable damage to the neighboring muscle and connective tissue.

3. Mixtures of proflavine and sulphapyridine powders when applied to brain tissue indicate a similar damaging effect of the proflavine. Mixtures containing only 0.5 per cent of proflavine sulphate are appreciably less damaging than those with higher percentage of this compound.

SOOY.

A Case of Aspirin Poisoning.

Charters, A. D.: Brit. M. J., Jan. 1, 1944, p. 10.

A patient is presented who recovered following the ingestion of 750 grains of aspirin with suicidal intent.

Signs consisted of drowsiness, disorientation, tinnitus, rapid deep respiration, rapid feeble pulse, and profuse sweating. A slight hepatomegaly was noticed on the fifth day.

Laboratory examination revealed a concentrated acid urine containing acetone, bile, glucose, albumin, erythrocytes, and leukocytes. The ferric chloride test for aspirin was strongly positive and remained so for four days.

Treatment consisted of gastric lavage with sodium bicarbonate solution, sodium bicarbonate gr. 30 given orally every two hours for the acidosis, high carbohydrate diet plus parenteral glucose and insulin for the ketosis and hepatitis.

The differential diagnosis from cardiac disease, ketosis, renal asthma, and infectious encephalitis is discussed.

SOOY.

Histaminic Cephalgia.

Swanson, L. W.: J. Allergy 15:144 (Mar.) 1944.

The author cites a case of histaminic cephalgia. The patient was a 37-year-old housewife who for several years had suffered from severe headaches at intervals of five to six weeks. These lasted for from several hours to an entire day, for as long as four weeks at a time. The headaches were unilateral, from the frontal to the occipital area, accompanied by injection of the conjunctival vessels and

swelling of the eyelids, and when severe and persistent, by nausea and vomiting. They were not relieved by ordinary analgesics or narcotics. A headache was activated by as little as 0.005 mg. of histamine injected intracutaneously, and was relieved by epinephrine. Because reinjection of histamine precipitated a succession of daily headaches, histaminase (15 units, three times daily) was given by mouth. Within three days all symptoms disappeared and had not recurred for four months. He calls attention to the fact that nausea and vomiting did occur notwithstanding Horton's statement to the contrary.

McMAHON.

Treatment of the Lip and Cheek in Cases of Facial Paralysis.

Dahlberg, A. A.: J. A. M. A. 124:503 (Feb. 19) 1943.

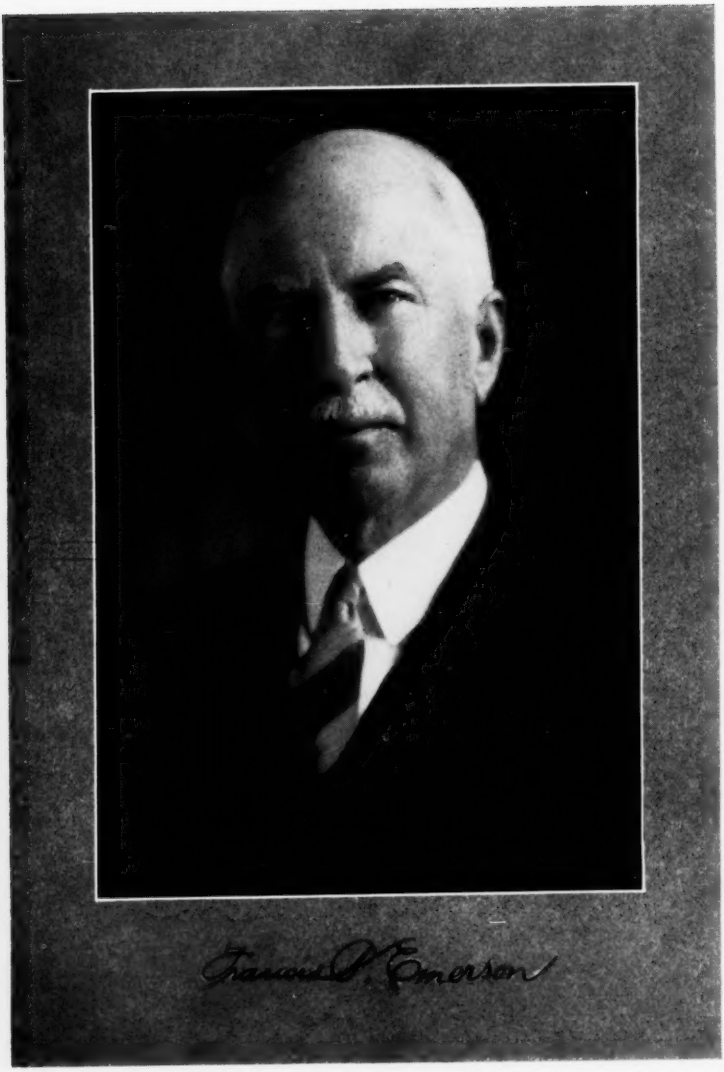
A dental device to keep the corner of the mouth elevated in cases of Bell's palsy is described.

It consists of a small hook anchored to an upper molar by means of a gold band, a small molded plastic hook which engages the corner of the mouth, and a connecting rubber band for tension.

Advantages claimed are inconspicuousness and lack of discomfort.

SOOY.

Obituary


Frank P. Emerson

FRANCIS P. EMERSON, M. D.

1862-1944

At his home in Franklin, Massachusetts, on January 19, 1944, Francis P. Emerson died at the age of 82 years. Of old New England ancestry, he was born at Candia, New Hampshire, June 10, 1862.

He was educated at the local country schools, by private tutors, and at Pembroke Academy in New Hampshire. He received his M.D. degree from the College of Physicians and Surgeons of Columbia University in 1886.

After five years of general practice in Roxbury, Massachusetts, he specialized in otolaryngology.

His wife, the late Rena Colby of Boston, died in 1932.

Dr. Emerson's entire medical life was spent in Boston where he was Oral Surgeon, later Consultant, to the Massachusetts Eye and Ear Infirmary and to the Massachusetts General Hospital; Consulting Oral Surgeon to the Adams Nervine Hospital, Jamaica Plains, and he was a member of the Staff of Brooks Hospital and of the Massachusetts Women's Hospital. For many years he was Instructor in Otology at the Harvard Medical School and also served on the Editorial Board of the Annals of Otology, Rhinology and Laryngology.

Membership in national special societies included that of the American Medical Association, serving as Chairman of the Section of Otology, Rhinology and Laryngology (1916-1917); the American Otological Society; the American Laryngological Society; the American Laryngological, Rhinological and Otological Society; The Massachusetts Medical Society; The American College of Physicians, and the New England Laryngological and Otological Society, serving as President in 1928. He was an Honorary Member of the Vermont State Medical Society and of the Rotary Club of Franklin.

Nonmedical activities included membership in the Washington Lodge, A. F. and A. M.; the Military Order of the World War; Boston Chamber of Commerce; The Harvard Club of Boston, the Weston Golf Club, and the Franklin Country Club.

During the first World War Dr. Emerson held the rank of Major, M.R.C., and was Chief of Head Surgery at Camp Lee, Petersburg, Virginia, during 1918 and 1919. During the influenza epidemic he

was Chief of the Emergency Hospital for influenza patients at Camp Lee.

He found recreation in golf and travel. Dr. Emerson was a man of great dignity, keen intellect and an inquiring mind. He early realized the importance of a knowledge of histopathology in otolaryngology. He was among the first to question the classical classification of deafness and its etiology. He was a firm believer in the theories of focal infection and many of his writings were on the relation of focal infection, not only to systemic disease but to chronic progressive deafness.

He is survived by a brother, Dr. William R. P. Emerson, and a daughter Margaret, now Mrs. Donald B. Chapman, of Franklin.

LeRoy A. Schall.

Publications of Dr. Francis P. Emerson

1. Rosenmueller's Fossae and Their Importance in Relation to the Middle Ear. Boston Med. & Surg. J. 48:544-547 (April 23) 1908.

2. General Lympho-Sarcoma Accentuated in the Pharynx. Laryngoscope, Feb., 1908.

3. The Responsibility of the General Practitioner and the Specialist in the Prevention of Deafness. Trans. New Hampshire Med. Society, 1909.

4. The Results of the Operation of Submucous Resection of the Septum in Private Practice. J. A. M. A. 55:1449-1450 (Oct. 22) 1910.

5. Chronic Focal Suppuration of the Head with General Symptoms, Especially in Adults. Boston Med. & Surg. J. 168:85-90 (Jan. 16) 1913.

6. Atrophic Rhinitis with Ozena—Its Etiology and Surgical Treatment. Annals of Otology, Rhinology and Laryngology 22:333 (June) 1913.

7. Vertigo of Labyrinthine Origin. Trans. Amer. Laryng., Rhin. & Otol. Society, 1915.

8. Changing Methods and Advances in the Treatment of Progressive Deafness from Chronic Secretory Otitis Media. Boston Med. & Surg. J. 177:583-588 (Oct. 25) 1917.

9. The Relation of Laryngology, Rhinology and Otology to General Medicine. J. A. M. A. 69:859-860 (Sept. 15) 1917.

10. Changing Methods and Advances in the Treatment of Progressive Deafness. (Second Communication). Boston Med. & Surg. J. 180:519-523 (May 8) 1919.

11. The Minor Role of the Conduction Apparatus in Slowly Progressive Deafness. Boston Med. & Surg. J. 183:736-741 (Dec. 23) 1920.

12. Clinical Manifestations of Infection of the Lateral Sinus. J. A. M. A. 75: 372-375 (Aug. 7) 1920.

13. Perception Deafness. Trans. Amer. Otol. Soc., 1921.

14. Polypoid Degeneration of the Lining of the Antrum of Highmore. Laryngoscope, May, 1921.

15. Indications for Opening the Mastoid Cortex. Boston Med. & Surg. J. 186: 301-303 (Mar. 9) 1922.

16. Report of Two Cases of Herpes Zoster Oticus with Special Reference to their Etiology. Laryngoscope, Feb., 1924.

17. Is Chronic Progressive Deafness a Rhinologic or Otologic Problem? ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 33:865 (Sept.) 1924.

18. The Gradual Change in the Otologists' Conception of the Conduction and Perception of Sound Impulses. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 40:710-716 (Sept.) 1931.

19. Has Science Thrown any New Light on our Understanding of Chronic Progressive Deafness? ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 50: 9-12 (March) 1931.

Notices

The American Board of Otolaryngology

The American Board of Otolaryngology will conduct an examination in Chicago at the Palmer House on October 4-7, 1944. Communications should be addressed to the Secretary, Dr. Dean M. Lierle, University Hospital, Iowa City, Iowa.

Second South American Congress of Otorhinolaryngology

The second South American Congress of Otorhinolaryngology will take place in Montevideo during the first half of October 1944. Seven speakers from as many countries have been designated as invited essayists.

The official speeches and all other communications must be in the hands of the General Secretary of the Organizing Committee before July 31 and August 31, respectively.

The registration fee is $12.00—Uruguayan pesos. Those who wish to participate should address the President of the Society of Otorhinolaryngology of his country or the General Secretary of the Executive Committee, Dr. Juan Carlos Oreggia, Yi 1491, Montevideo, Uruguay.

Biological Photographic Association

The Biological Photographic Association will hold its Fourteenth Annual Meeting on September 7, 8 and 9 at the Arlington Hotel, Binghamton, New York. Information regarding the Association and the convention program may be obtained from the Secretary, University Office, Magee Hospital, Pittsburg 13, Pa.

University of Iowa Summer Session

The State University of Iowa will feature in its 1944 Summer Session an intensive four-weeks course in Audiometry and Fitting of Hearing Aids from June 26 to July 22. Running concurrently with this course will be a Conference Series on Speech and Hearing Rehabilitation each weekend from June 23 to July 22.

The Summer Speech Clinic for school children and adults will be held from June 19 to July 28. In addition to these special courses the regular program of graduate study leading to the M.A. and Ph.D. degrees in speech pathology or hearing conservation will be offered.

For further information address Dr. Dean M. Lierle, University Hospital, Iowa City, Iowa.

Northwestern University, School of Speech

The School of Speech of Northwestern University presents a Symposium in Hearing Aids and Residual Hearing, offered as part of the Program in the Education of the Deaf and Hard of Hearing, at the 1944 summer sessions, June 26 through August 26. For further information write to James H. McBurney, Dean, School of Speech, Northwestern University, Evanston, Illinois.

University of Illinois

The University of Illinois College of Medicine announces its fall didactic and clinical course for specialists in otolaryngology from September 25 to 30 inclusive. Registration is limited to 25. For information please write to Department of Otolaryngology, University of Illinois College of Medicine, 1853 W. Polk Street, Chicago 12, Illinois.

Directory of Medical Specialists

The third edition of the Directory of Medical Specialists listing names and biographic data of all men certified by the fifteen American Boards is to be published early in 1945. Diplomates are requested to make prompt return of the notices regarding their biographies as soon as possible after receiving the proper forms soon to be mailed from the publication office.